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(See inside of back cover)

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THE PRINTING OF THIS PUBLICATION HAS BEEN APPROVED BY
THE DIRECTOR OF THE BUREAU OF THE BUDGET, 20 FEB. 1956

Monthly Message

We are very happy to announce the formation of a Joint Committee on Aviation Pathology, a most important group. The establishment of this Committee required some several months inasmuch as it is a joint committee of the governments of Great Britain, Canada, and the United States. Although the Air Force of each country was fully in favor of this Committee and had met once or twice informally, in order to bring about maximum fulfillment of the possible functions, it was necessary to reach a formal agreement among our three countries. This has been accomplished. As many of you may not know, the first clue to the cause of the two Comet disasters in the Mediterranean over a year ago was by means of the ordinary autopsy which revealed evidence of explosive decompression in each body. An excellent report of this medical intelligence appeared in *Lancet* in June 1955 and this in turn was picked up by our popular magazine *True* in August 1955.

I wish to acknowledge my personal thanks to Wing Commander Bruce Harvey of the RAF, Wing Commander R. H. Lowry, CAF, and Dr. Howard Karsner, Consultant in Medical Research in the U. S. Navy, for their vision in realizing the need for this Committee and all that they have done in its organization. We fully expect that the recommendations and findings of the Committee will contribute greatly to our knowledge of the causes of fatal airplane accidents, in which pilot failure for one reason or another is too frequently a primary factor.

Frank B. Berry

FRANK B. BERRY, M. D.
Assistant Secretary of Defense
(Health and Medical)

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Foreword

The *United States Armed Forces Medical Journal* is the medium for disseminating information of administrative and professional interest to all medical personnel of the Department of Defense. The Assistant Secretary of Defense (Health and Medical) and the Surgeons General of the several services invite all medical officers, dental officers, Medical Service Corps officers, Nurse Corps officers, and officers of the Veterinary Corps of the Armed Forces, and the medical consultants of the Army, Navy, and Air Force to submit manuscripts for publication in this *Journal*.

FRANK B. BERRY, M. D.,
Assistant Secretary of Defense (Health and Medical).

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UNITED STATES ARMED FORCES
MEDICAL JOURNAL

Volume VII July 1956 Number 7

COMMON RESPIRATORY DISEASE IN ARMY RECRUIT POPULATIONS

An Epidemiologic Study

FRATIS L. DUFF, *Colonel, USAF (MC)*

THE GREATEST single cause for admission of armed services personnel to medical treatment facilities is a group of acute diseases affecting the upper respiratory tract which, in military morbidity reports, are grouped under the term "common respiratory disease." Even though only a brief period of disability is experienced, common respiratory disease produces more time lost from duty than any other cause except injuries. Approximately six per cent of all days lost from duty is due to these conditions.[1,2]

The greatest impact made by common respiratory disease upon military populations has been during periods of mobilization for war. Young adults mobilized into the military forces of the United States are particularly susceptible to these diseases during the first months of military service. Epidemics of common respiratory disease frequently occur when large numbers of these recruits are concentrated in military training camps.[3-5]

During the period of World War II (1942-1945), from a mean strength of 6,076,000 there were 4,086,000 admissions for common respiratory disease recorded by the U. S. Army. The average time lost from duty by a person admitted for treatment of common respiratory disease during this period was 6.5 days.[6] These four million cases during the war period thus resulted in approximately 265 million man-days lost from duty, or an average of approximately 18,000 persons absent from duty each day of the war.

From U. S. Air Force School of Aviation Medicine, Gunter Air Force Base, Ala.

Review of the total Army experience with common respiratory disease during the mobilization and war period (fig. 1) reveals that the highest reported incidence was during the winter of 1940-1941, when the National Guard divisions were being mobilized.[7] Similar epidemics of lesser magnitude occurred during the following three winters. The sharp epidemic during the winter of 1943 was due to influenza which appeared in November of that year in both civilian and military populations and rapidly spread over the United States, causing explosive epidemics in many Army camps.[8,9] Following this epidemic, the incidence of common respiratory disease underwent a marked and progressive decline throughout the remainder of the war.

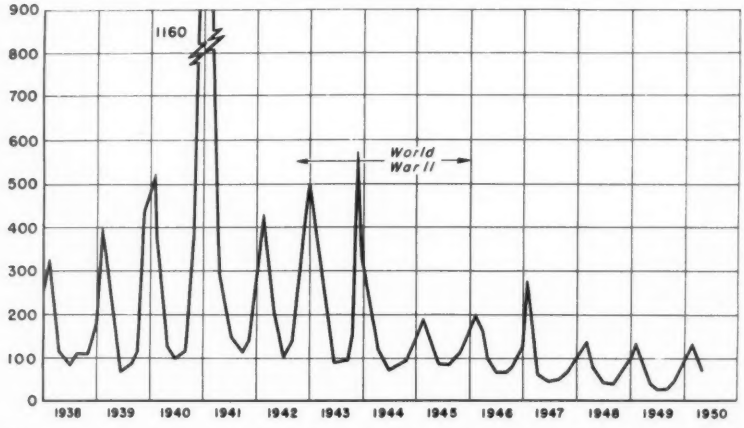

Figure 1. Common respiratory disease and influenza from 1938 to 1950 (admission rates per 1,000 troops per year) from monthly statistical health reports.

One element of the composition of the Army that closely paralleled the incidence of common respiratory disease was the proportion of recruits to the total number of Army troops.[7] These data revealed a positive correlation between the percentage of enlisted men with less than six months' service and the incidence of common respiratory disease. The epidemics experienced during the mobilization period and the first two years of the war occurred when a large proportion of Army personnel was composed of recruits, and the marked reduction in incidence occurred when a very small proportion of Army troops consisted of recruits. It would appear that the high common respiratory disease rates reported during the first years of the war were in large part due to epidemics among recruits.

A study of the experience of U. S. Army troops with common respiratory disease in the various theatres of operation revealed

outstanding differences in the monthly incidence reported from the various geographic areas.[7]

In the Alaskan, North American, and European Theatres of Operation the incidence increased markedly during the winter season, frequently to epidemic proportions, and the total four-year rate was high. In the tropical and subtropical areas of Latin America and the Southwest Pacific and Pacific Ocean Theatres there was an almost complete absence of seasonal variation, and the rate remained at a very low level throughout the war period.

Common respiratory disease among Army troops in the continental United States was a problem of major importance, the total rate for the four-year period being 192 cases per 1,000 troops per year.

The variation in seasonal peaks and total incidence observed in the northern and southern overseas theatres was also apparent in the admissions reported from geographic subdivisions or service commands in the continental United States. The highest incidence and the greatest increases during the winter months were observed in the Second, Fifth, Sixth, and Seventh Service Commands, composed of the northern and northeastern states. The lowest incidence and the smaller seasonal variations were observed in the southern and southwestern states of the Fourth, Eighth, and Ninth Service Commands.[7]

These data suggest that there is a factor or factors in the northern overseas areas and in the northern part of the United States which apparently enhances the transmission of common respiratory disease and that this element is less prevalent or less effective in the southern areas of the United States and almost completely absent in the tropical and subtropical overseas areas.

Several carefully controlled studies conducted during the war period have demonstrated that recruit populations experienced severe epidemics of common respiratory disease if assembled during the winter months.[4, 5, 10] Sartwell,[3] in a study of recruits at Fort Dix, N. J., conducted after the war, observed severe epidemics among recruit units assembled in January and February. Units formed in the fall and early winter did not experience severe epidemics early in the training period or during the following January or February, when the newly arrived units were having sharp epidemics. These studies and others[7] demonstrate that recruit units show marked increases in common respiratory disease rates during the first weeks of military service and that severe epidemics are experienced by such groups if they are assembled during the winter season.

There are, therefore, indications that recruit units assembled during a season of low common respiratory disease incidence do not experience epidemics at that time or during subsequent winter seasons. This effect has not, however, been clearly demonstrated since the period of observation in the basic training centers has not been of sufficient duration to follow these recruit groups throughout the entire year.

There apparently is a "seasoning" process which operates in recruit units to make the personnel more resistant to common respiratory disease, as measured by admissions to medical treatment facilities. It has not been demonstrated whether this seasoning is dependent upon the individual's having experienced the disease or is accomplished by the intimate intermingling of recruits and incident to the increasing length of service and adjustment to military life. It has not been clearly demonstrated whether or not recruit groups may become seasoned without experiencing extensive epidemics of common respiratory disease and enormous loss of manpower from vital training duties. In order to answer these questions it is necessary that recruit populations be observed for periods of time longer than those of previous studies. These observations should extend throughout the first year of service.

The previous studies have been made on different recruit units, all of which were assembled in the same geographic location. No comparisons have been made between recruit units in different geographic areas during the same season so as to determine the effect of geographic location as well as season upon recruit epidemics. Observations should therefore be of recruit populations assembled in various seasons of the year and in different geographic locations during the same season of the year.

It appeared that the Army divisions of World War II were large recruit populations that could be followed from time of assembly through the various phases of military service. Army divisions are tactical organizations composed of approximately 15,000 individuals. They were assembled in different geographic areas throughout the United States for training and then deployed to theatres of operation.

These divisions were of three different types: Regular Army divisions, National Guard divisions, and Army of the United States or Organized Reserve divisions.

Prior to the war there were seven incompletely manned Regular Army divisions. Seven additional divisions were activated during 1940 and 1941, and the old divisions brought up to strength. The new divisions were formed with cadres of varying sizes from

the old divisions, and then were brought up to strength with personnel primarily from the reception centers.[11]

The National Guard divisions existed prior to the war as state organizations. Units of the divisions were organized by the cities and towns throughout the state or states. The units assembled weekly in the local community for training purposes, and the division as a whole assembled once a year in the summer for two weeks of field training. The National Guard divisions were called into the Federal service during 1940 and 1941. Personnel from the local units were rapidly assembled as a division and then moved to an Army camp for training. Necessary filler personnel were supplied from recruit stations or induction centers. The National Guard divisions were large recruit populations with all personnel having the same length of military service. The personnel of the division came directly from civilian life and from the same geographic area, *i. e.*, the same state or adjacent states.

The Army of the United States and Organized Reserve divisions were activated during 1942 and 1943.[11] These divisions were created according to a plan which was developed in January 1942.[12] Each new division was assembled around a cadre of 159 officers and 1,190 enlisted men drawn from the older divisions or from replacement training centers. The bulk of the troops (13,425 enlisted personnel) came into separate divisions over a period of two to three weeks, directly from reception centers.[12]

Premobilization plans had contemplated replacement training centers as an integral part of the process of mobilization. These centers were designed to provide basic training of three to four months' duration for all persons before their assignment to divisions. With the declaration of war in December 1941, it was decided not to expand these centers commensurate with the great expansion of the Army. Graduates from the replacement training center were used as replacements for units overseas or alerted for overseas and as cadres for new divisions. In many instances the flow through these centers was insufficient to fulfill these requirements.[12]

Filler personnel for new divisions came directly from reception centers. These centers were located in various areas of the United States, and received inductees from civilian life for processing. In the induction center the recruits received administrative processing, uniforms, and assignment to a training unit or to a newly activated division. The men were usually retained in the induction center less than one week. Thus the newly activated divisions were composed primarily of recruits coming from many different areas of the United States.

This study was conducted on the experience of these Army divisions with common respiratory disease during World War II in order to demonstrate certain epidemiologic features of these diseases in large military recruit populations. A primary aim of the study was to demonstrate those epidemiologic features which might be useful in the development of practical methods for the reduction of common respiratory disease in military populations, particularly during periods of rapid mobilization.

SOURCE OF DATA

A search through the files of the Adjutant General's Historical Record Depot revealed medical records complete through the first year of service for 19 divisions. The incidence of common respiratory disease has been followed each month from the time of assembly through the first year, and for some units throughout the war period. The admissions to hospital or quarters for common respiratory disease and the mean strength have been obtained from the original weekly Statistical Health Report. The data from these reports are a reliable guide to the trends in morbidity and may be used for comparison with other data from the same source.[5,7] The monthly incidence is expressed as admissions per 1,000 troops per year.

Frequently there were delays of varying length from the date of the official activation order until the filler personnel of the unit were assembled. Therefore, the study of each division is dated from the time of the assembly of the personnel rather than from the date of the official activation order.

The term "common respiratory disease" is used in the Army, and has been used in this study, to designate those diseases of the upper respiratory tract for which no causative agent is identified. A great number of diagnostic terms are used to describe these conditions. The most frequently used are acute catarrhal nasopharyngitis, acute catarrhal pharyngitis, acute catarrhal bronchitis, acute laryngitis, common cold, and acute respiratory infection, all of which are grouped together in Army morbidity reports as common respiratory diseases.

Those cases diagnosed as influenza are reported separately. In the compilations made by the Surgeon General's Office and in this study, influenza is included with common respiratory disease because of the confusion and lack of definitiveness which arise in the clinical diagnosis of this condition during nonepidemic periods and the tendency to diagnose most acute respiratory diseases as influenza during epidemic periods.

The incidence data used in this study are calculated from the cases of men admitted to hospital or quarters for treatment. Studies conducted by the Commission on Acute Respiratory

Diseases of the Army Epidemiological Board demonstrated an increased incidence during late summer and fall of a mild respiratory disease usually treated in the dispensary without hospitalization.[13,14] The epidemiologic pattern of this minor respiratory disease differed from that of the more severe respiratory diseases necessitating hospitalization. The more severe respiratory diseases of unknown cause commonly producing large numbers of hospitalizations in recruit populations were called "undifferentiated acute respiratory disease" and commonly referred to as "ARD" by the Commission on Acute Respiratory Disease in their studies during World War II.[5] It would appear that a large proportion of the cases of acute respiratory disease in recruit populations necessitating hospitalization and reported under the Army classification of common respiratory disease was of the type studied by the Commission and termed ARD.

Recently there have been reports of isolation of a new group of viruses that are closely associated with the type of acute respiratory disease called ARD.[15,16] The isolation of these agents gives some hope of identification of the causative agent of the acute respiratory illness which commonly produces epidemics in military recruit organizations. The identification of this agent will make possible more detailed studies and enhance the development of control measures.

EPIDEMIOLOGIC OBSERVATIONS

Divisions Assembled in Winter of 1940-1941

Records are available on four National Guard divisions assembled during the winter of 1940-1941. The 35th Division assembled in January 1941, the 26th Division assembled in February, the 33d Division in March of that year, and the 30th in October 1940. The monthly incidence of common respiratory disease and mean strength for these units is shown in figures 2 and 3.

The 35th Division was composed of troops from Kansas, Missouri, and Nebraska. Troops from these three states were assembled at Lincoln, Nebr., in January 1941 and then moved to Camp Robinson, Ark., for training.

At the time of the initial assembly of the division and during the build-up to full division strength in January and February, the unit experienced common respiratory disease in epidemic proportions. The initial medical report revealed rates of 3,100 per 1,000 troops per year. This excessive incidence of respiratory disease decreased rapidly during the first four months and after the unit became stabilized at Camp Robinson, Ark., the rates were approximately 200. This unit did not again experience an epidemic of common respiratory disease. The seasonal in-

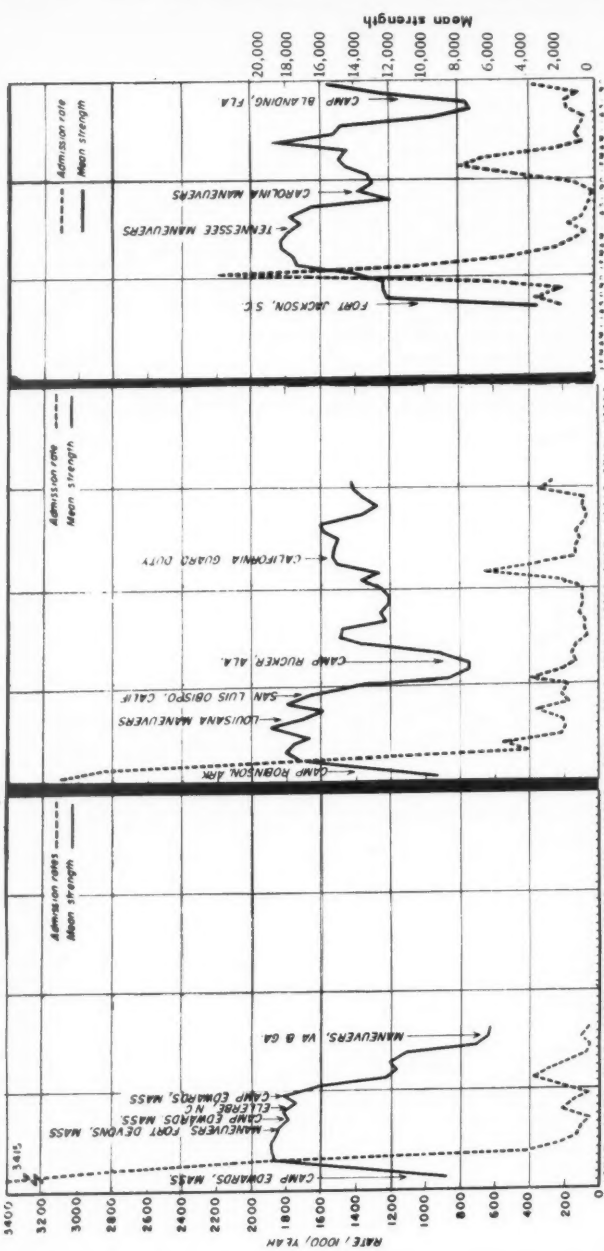

Figure 2 Admission rates for common respiratory disease and influenza and monthly mean strength for three divisions assembled during the winter of 1940-1941.

creases in the unit reported during the subsequent winter seasons were less than the rates reported by total Army troops in that area. The movement of the division to the Louisiana maneuver areas, and later to California, produced no marked effect upon the common respiratory disease rates. The marked reduction in strength during early 1942 was due to the formation of cadres for the new divisions being activated at that time. The replacement of these losses during the summer months at Camp Rucker, Ala., produced no marked effect upon the incidence of common respiratory disease reported at that time or during the subsequent winter months.

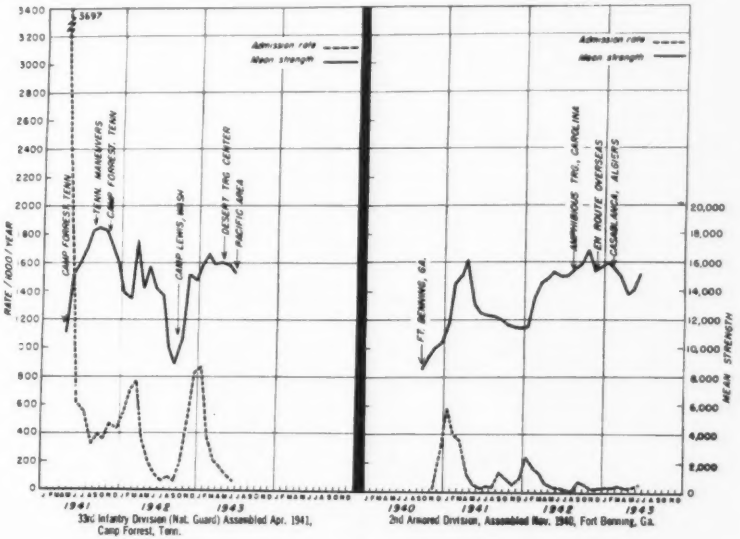

Figure 3. Admission rates for common respiratory disease and influenza and monthly mean strength for two divisions assembled during the winter of 1940-1941.

The other National Guard divisions assembled in the northern states showed similar epidemic patterns. The 26th Division, Massachusetts National Guard, was assembled at Camp Edwards, Mass., in February 1941. This unit also had a severe epidemic when first assembled, with a rate of 3,415 per 1,000 troops per year reported at that time. After this epidemic the common respiratory disease rates remained at a low level with only a small seasonal increase during the second winter. This unit moved several times during the latter part of the first year of service—to the Massachusetts maneuver area, to North Carolina, and back to Massachusetts. Medical records are available only for the first year and a half of service.

The 33d Division was composed of troops from the state of Illinois and was assembled at Chicago, Ill., in March 1941. It should be noted that this area was in the Sixth Service Command, which reported the highest common respiratory disease rates for the war period.

At the time of assembly the troops of the unit experienced a very severe epidemic, reporting a rate of 3,697 per 1,000 troops per year. This unit reported higher seasonal increases of common respiratory disease in subsequent winters than did the other two units, although these increases were approximately the same as those reported by all Army troops in that area. These winter peaks occurred at the time the unit received large numbers of filler personnel as replacements for cadres furnished to other divisions. The effect of the addition of new recruits to organizations of seasoned men in increasing respiratory disease rates was observed by the Commission on Acute Respiratory Diseases in their studies at Fort Bragg.[5] This Division moved from Camp Forrest, Tenn., to the Tennessee maneuver area, and during the second year of activation to Fort Lewis, Wash., to the Desert Training Center, and then to the Pacific area.

The 39th National Guard Division, with troops from North and South Carolina, Georgia, and Tennessee, was assembled at Fort Jackson, S. C., from October 1940 through January 1941. The assembly of this recruit population at this time produced an epidemic of common respiratory disease with a peak rate of 2,174 per 1,000 troops per year being reported in January 1941. This epidemic was of short duration, and the rate fell to a low level and then followed the pattern observed in the other divisions. This unit trained at Fort Jackson, S. C., and the Tennessee and Carolina maneuver grounds, and then moved to Camp Blanding, Fla. This division, assembled in the winter season but in a more southern state, experienced a typical recruit epidemic but of much lower magnitude than the National Guard divisions assembled in the northern states.

Records are available for the Second Armored Division, which was assembled at Fort Benning, Ga., in November 1940. This unit was built up rapidly from a cadre to full division strength during December 1940 and January of 1941. This rapid influx of personnel produced an increase in the incidence of common respiratory disease to a rate of approximately 600 per 1,000 troops per year. Following this minor epidemic the incidence of common respiratory disease in this unit remained at a very low level for the next two years.

This unit is not strictly comparable to the National Guard divisions as the personnel were not all recruits from the same geographic area, and the division was activated in a different

manner. The unit did, however, increase 100 per cent in strength by the addition of filler personnel during the build-up phase. This unit, assembled in Georgia, did not experience an epidemic of common respiratory disease at the time of assembly of the troops or during the two subsequent winter seasons. The movement of the unit to other areas in the United States and overseas had no marked effect upon the common respiratory disease rate.

Divisions Assembled in Summer of 1942

Medical records are available on five divisions assembled during the summer of 1942 (figs. 4 and 5). The 89th Division was activated at Camp Carson, Colo., the 95th Division at Camp Swift, Tex., the 81st Division at Camp Rucker, Ala., the 77th Division at Fort Jackson, S. C., and the 85th Division at Camp Shelby, Miss.

Patterns of the monthly common respiratory disease incidence in the divisions assembled in Texas, Alabama, and South Carolina were very similar. These units showed only slight increases in incidence at the time of assembly of the personnel. Subsequent winter seasons were characterized by seasonal increases of low magnitudes.

The 95th Division was assembled in July at Camp Swift, Tex., and trained in Texas, Louisiana, and the Desert Training Center. This unit then moved to Indiantown Gap, Pa., in January and February of 1944 and after a period of training was shipped to England. The movement into a northern state during the winter season did not cause an appreciable increase in the reported incidence of common respiratory disease.

The 81st Division was assembled in June at Camp Rucker, Ala., and spent the first year training at this location. The unit then moved to the Tennessee maneuver area, to the Desert Training Center, and later to California. These movements had no effect upon the reported incidence of common respiratory disease.

The 77th Division was assembled at Fort Jackson, S. C., in March 1942. The increase in common respiratory disease at the time of assembly was much less than the increase noted in the 30th Division assembled at the same place during the preceding winter months. This unit trained in the Louisiana maneuver area, the Desert Training Center, and Camp Pickett, Va., and was then shipped to the Pacific area. Medical records are available for the period that the unit was in Okinawa, regrouping in the Philippines, and entering Japan for occupation duty. During these periods of training and combat duty, seasonal increases of low magnitude in common respiratory disease were reported.

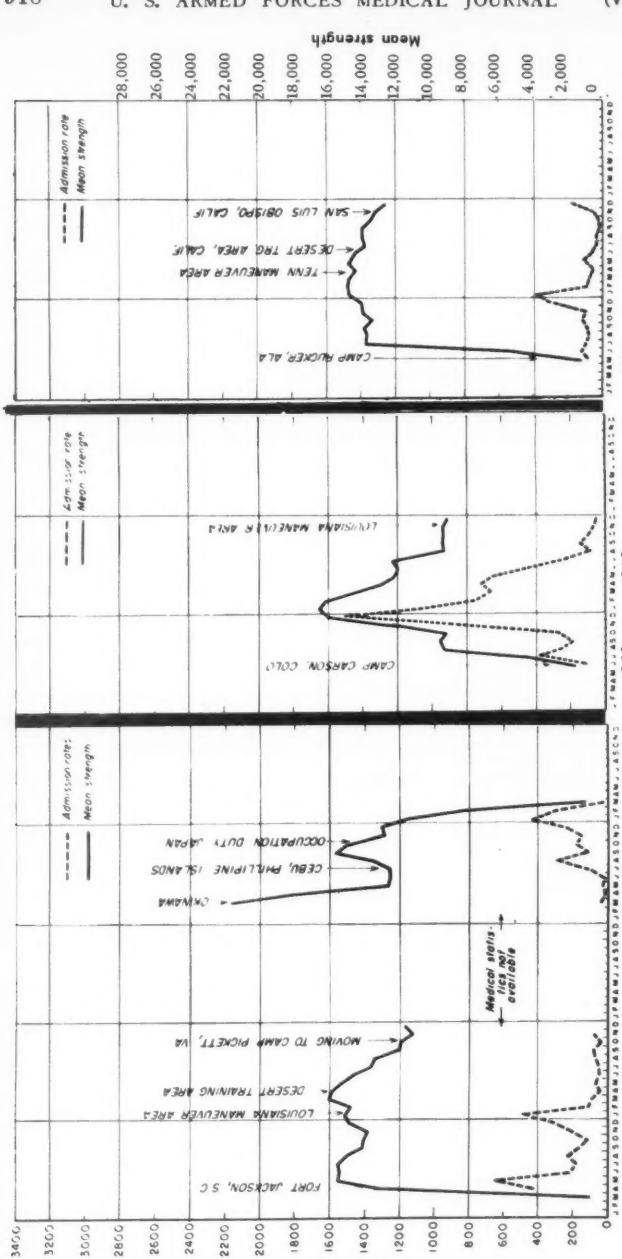

Figure 4. Admission rates for common respiratory disease and influenza and monthly mean strength for three divisions assembled during the summer of 1942.

The 89th Division was assembled in July 1942 at Camp Carson, Colo. This unit was initially formed by approximately 9,000 personnel during July and August. A small increase in the common respiratory disease incidence was observed at that time. During November and December the unit was brought to full division strength of approximately 16,000 personnel by the addition of recruits. At this time there was a marked increase in common respiratory disease, and rates in excess of 1,500 per 1,000 troops per year were reported. These rates did not show the rapid decline that was observed in recruit epidemics of other units, and high rates were reported through July of the following summer. An appreciable amount of scarlet fever was reported from the unit during this period.

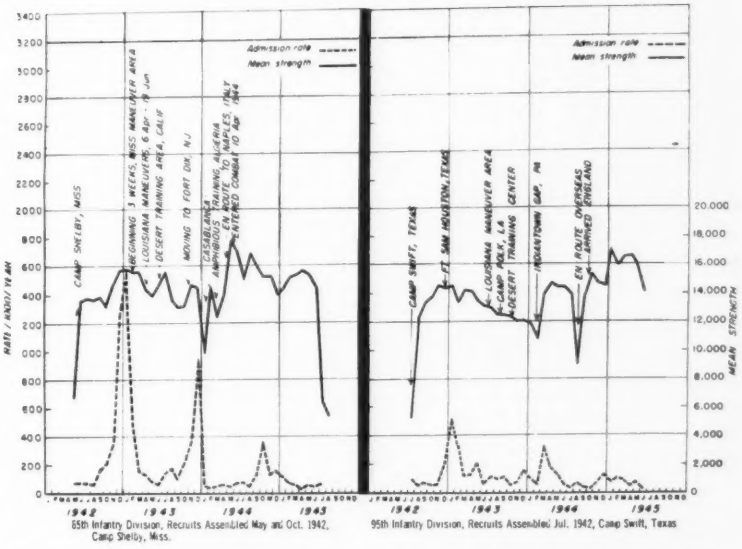

Figure 5. Admission rates for common respiratory disease and influenza and monthly mean strength for two divisions assembled during the summer of 1942.

The epidemic pattern of this unit was influenced by two factors. Army units stationed in Colorado during and after the war have consistently reported excessively high rates of streptococcal disease.[6] Characteristically, the high incidence of these diseases has been prolonged into late spring or early summer. This disease reported as common respiratory disease may have accounted for the prolongation of the high rates which produced an excessive yearly rate for the first year of training. This is suggested by the occurrence of scarlet fever in the unit.

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The sharp epidemic reported during November and December was influenced by the assignment of approximately 6,000 recruits to this unit at that time. Medical records for the second winter following activation were not available. The rates for December of the second winter, however, were low.

Medical records of the 85th Division also demonstrate the effect of the addition of a large number of recruits to a unit during the winter months. This unit was formed by the assembly of approximately 13,000 personnel in May at Camp Shelby, Miss. No increase in common respiratory disease was reported during the summer months. During October and November approximately 3,000 inductees entered the division and a sharp epidemic occurred in December and January. The peak incidence in the second winter season was influenced by influenza present at that time but was approximately the same as that reported by total Army troops in the area in which the division was stationed.

Three divisions which assembled during the summer months in the southern part of the United States did not experience recruit epidemics during the first few months of training or during the subsequent winter season, even after moving to less favorable geographic areas.

Two of these divisions had two periods during which the personnel were assembled. The initial formation of the division during the summer months did not produce epidemics of common respiratory diseases. The addition of large numbers of inductees to those units during the winter months did, however, produce sharp epidemics. The high incidence in the unit assembled in Colorado was prolonged into the summer months, thus producing a high rate during the first year of service.

Divisions Assembled in Winter of 1942-1943

Records are available from six divisions activated during the winter of 1942-1943 (figs. 6-8).

The 94th Division was officially activated at Camp Custer, Mich., in November 1942 and almost immediately moved to Camp Phillips, Kans., where most of the personnel were assembled in November and December. Common respiratory disease rates rose in epidemic manner to about 2,400 per 1,000 troops per year within the first three months and then fell to a low level. A seasonal rise of lesser magnitude was experienced during the winter of 1943-1944. This unit trained in the Tennessee maneuver area and then at Camp McCain, Miss.

The 83d Division was assembled during October and November 1942 at Camp Atterbury, Ind. A typical recruit epidemic with rates of approximately 2,000 occurred at that time. This epidemic was of short duration and the common respiratory disease

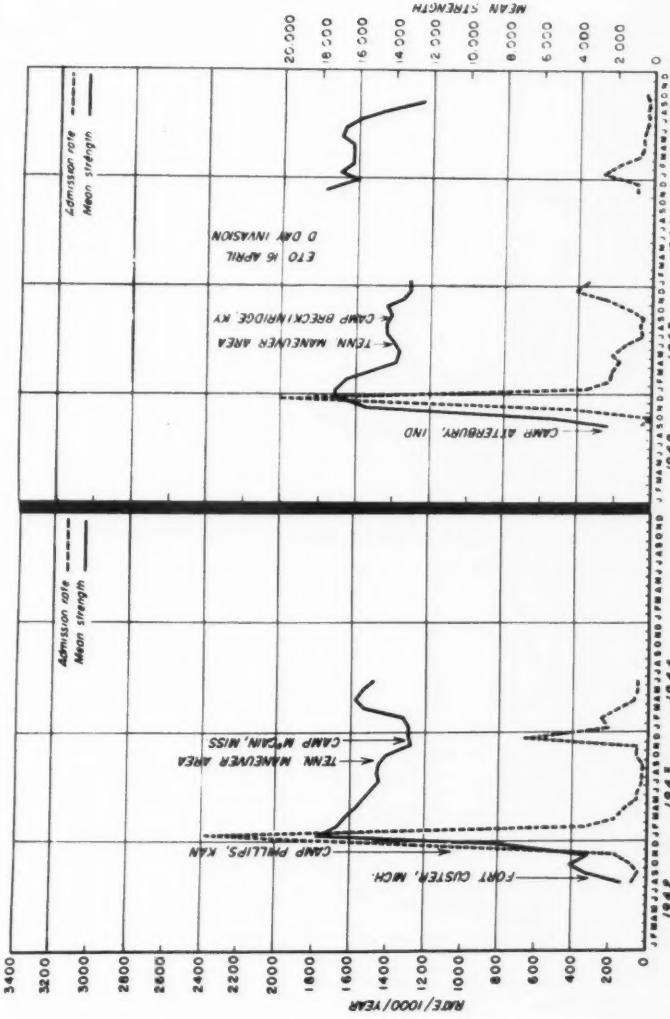
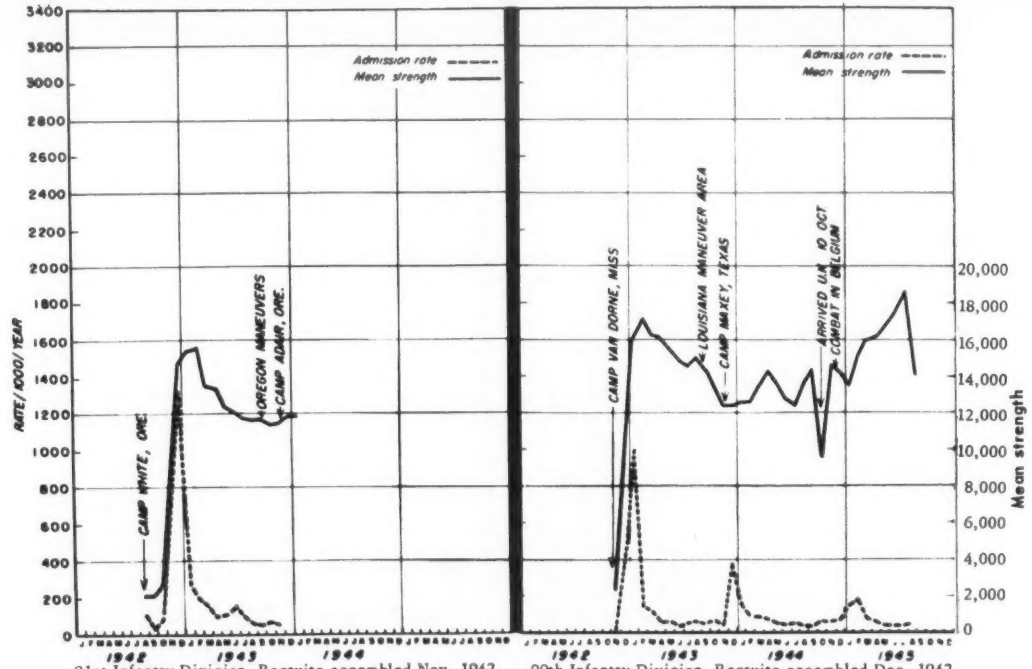


Figure 6. Admission rates for common respiratory disease and influenza and monthly mean strength for two divisions assembled during the winter of 1942-1943.



91st Infantry Division, Recruits assembled Nov. 1942, Camp White, Oreg.

99th Infantry Division, Recruits assembled Dec. 1942, Camp Van Dorne, Miss.

Figure 7. Admission rates for common respiratory disease and influenza and monthly mean strength for two divisions assembled during the winter of 1942-1943.

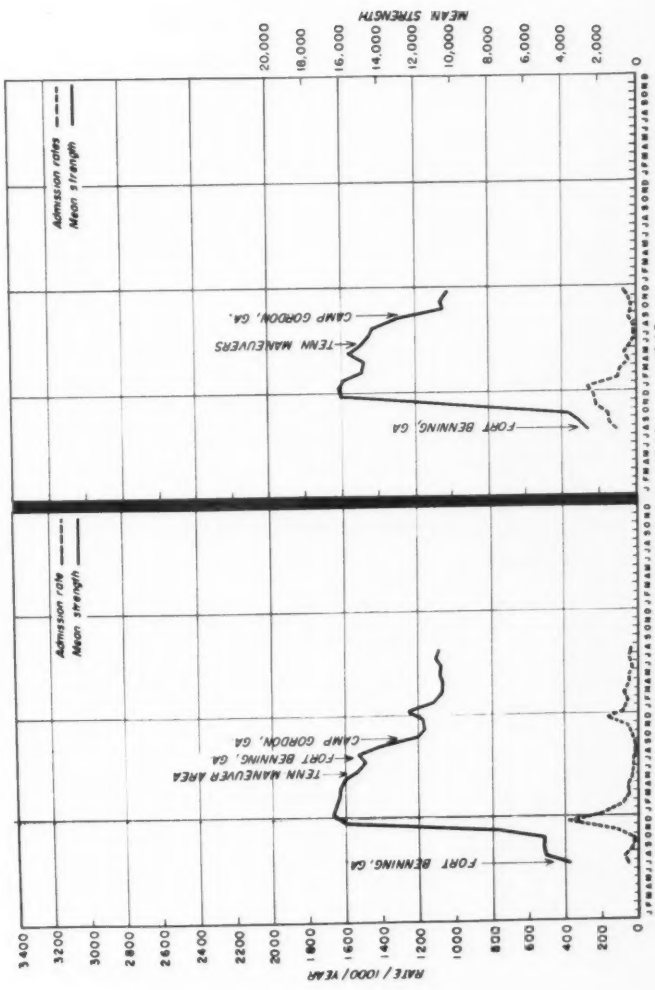


Figure 8. Admission rates for common respiratory disease and influenza and monthly mean strength for two divisions assembled during the winter of 1942-1943.

rates decreased to a low level throughout the remainder of the first year following activation. During the winter of 1943-1944 a normal seasonal increase was recorded. This unit trained at the Tennessee maneuver area and Camp Breckenridge, Ky., and was then shipped to the European Theatre of Operations. Medical records are not available for the period during the movement to England and during the invasion of the continent. During the winter of 1944-1945 when the division was engaged in field operations on the European continent a minor seasonal rise of respiratory disease was reported.

The 91st Division assembled at Camp White, Oreg., in November 1942. The strength increased very rapidly during this month and there occurred a marked increase in common respiratory disease with rates of approximately 1,300 per 1,000 troops per year during December. Following this epidemic, low rates were sustained throughout the remainder of the first year of service. The unit participated in the Oregon maneuvers and then moved to Camp Adair, Oreg., in the fall of 1943.

The 99th Division was initially formed at Camp Van Dorne, Miss. The personnel were assembled in November and December 1942. An increase in common respiratory disease was reported at that time with a rate of approximately 1,000 per 1,000 troops per year during January 1943. The rate fell rapidly to a very low level, which was maintained throughout the summer months. A normal seasonal increase was reported during the subsequent winter season. This unit trained in Mississippi and Texas and then moved to the European Theatre and into combat in Belgium. These movements produced no increase in the common respiratory disease rates.

Two divisions, the 10th and 11th Armored Divisions, were both activated at Fort Benning, Ga., and have practically the same epidemic patterns. These units were both assembled in November and December 1942. At this time a minor increase in common respiratory disease incidence was reported. During the second winter, while these units were in training, only small seasonal increases occurred. It is of interest that these two units, assembled at the same time and in the same geographic area, demonstrated an almost identical experience with common respiratory disease.

Of the six divisions assembled during October, November, and December 1942, two were assembled in northern states. These units, assembled in Kansas and Indiana, experienced marked epidemics of common respiratory diseases during the first three months of service. The three divisions assembled in Mississippi and Georgia during the same season did not experience epi-

demics. The unit assembled in Oregon reported common respiratory disease rates between these two extremes.

Divisions Assembled During 1943

Records are available for three divisions assembled during 1943 (fig. 9). The 106th Division assembled during March at Fort Jackson, S. C.; the 63d Division assembled in August and September at Camp Van Dorne, Miss.; and the 16th Armored Division assembled during July at Camp Chaffee, Ark. These units were all formed in southern states; records are not available for units formed in the north at this time.

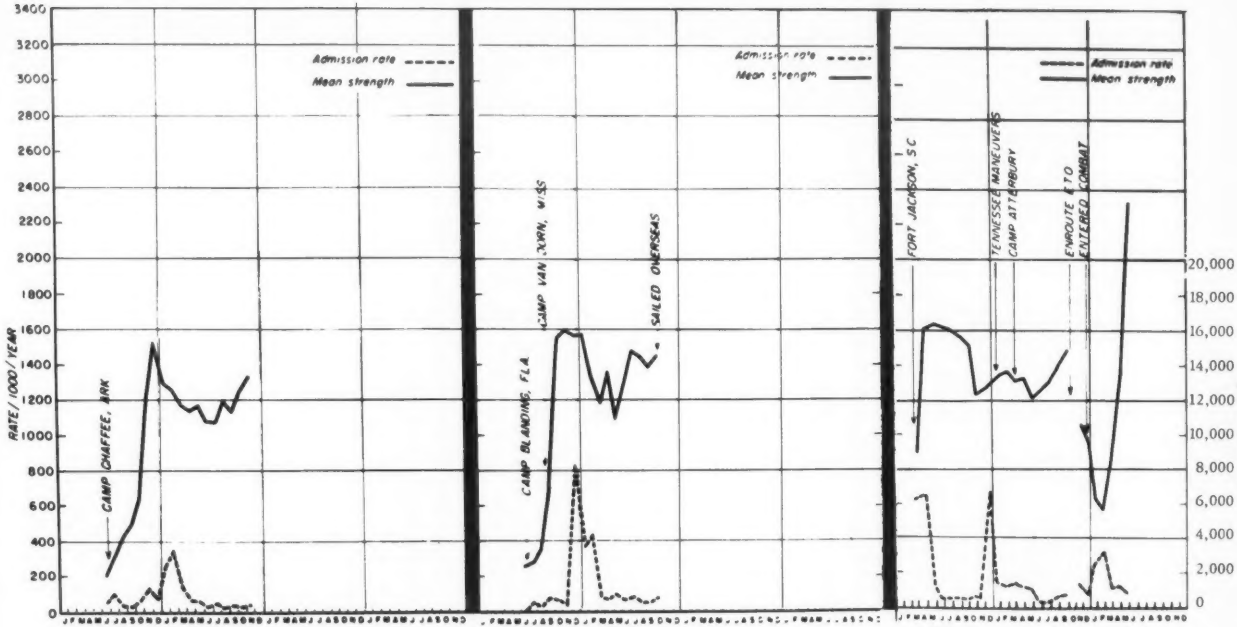
These units all exhibit epidemic patterns similar to those observed in other units assembled in the southern states in that slight to moderate increases in the incidence of common respiratory disease occurred at the initial assembly of the troops. During the subsequent winter season, moderate seasonal peaks comparable to those of all Army troops in that area were reported.

FINDINGS

Table 1 shows the common respiratory disease rates of the 19 Army divisions assembled during different seasons and in various geographic areas. The total respiratory disease rates for the first three months, the first year, and the second year of military service are tabulated.

These data and the charts of the divisions show that the units assembled during the winter seasons of 1940-1941 and 1942-1943 experienced high common respiratory disease rates during the first three months of service. Of these units, those assembled in the northern states reported severe epidemics while those assembled in southern states reported increases in common respiratory diseases of lesser magnitude. The total incidence of these diseases during the first year is influenced greatly by the magnitude of the recruit epidemics. Divisions assembled in the winter season in northern states reported high incidence for the first year, while the total first year's incidence for those assembled in southern states was low.

Divisions completely assembled during the summer months did not experience recruit epidemics at that time or during the subsequent winter season. The total incidence for the first year of service was low. Two divisions, the 85th and the 89th, experienced moderate epidemics with the addition of large numbers of recruits during the winter months. Those epidemics produced a high rate for the first year in these two units. The prolongation of high rates into the summer months and a high noneffectiveness from common respiratory disease during the first year occurred in the 89th Division assembled in Colorado.



16th Armored Division, Recruits assembled July 1943, Camp Chaffee, Ark.

63d Infantry Division Recruits assembled Sept. 1943, Camp Van Dorne, Miss.

106th Infantry Division, Recruits assembled Mar. 1943, Ft. Jackson, S. C.

Figure 9. Admission rates for common respiratory disease and influenza and monthly mean strength for divisions assembled in 1943.

TABLE 1. *Common respiratory disease in U. S. Army Divisions**

Period when divisions assembled	Division	Assembled		Common respiratory disease rate (on annual bases)		
		Month	Place	1st 3 mo	1st yr	2d yr
Winter of 1940-1941	26th	Feb.	Mass.	2,461	604	177
	35th	Jan.	Ark.	2,329	694	123
	33d	Apr.	Tenn.	1,689	751	344
	30th	Oct.	S. C.	377	448	239
	2d	Nov.	Ga.	366	195	73
Summer 1942	77th	Mar.	S. C.	449	271	104
	89th	Jul.-Nov.	Colo.	253	710	125
	81st	Jun.	Ala.	82	132	52
	85th	May-Oct.	Miss.	71	430	174
	95th	Jul.	Tex.	69	165	125
Winter of 1942-1943	94th	Nov.	Mich.	1,466	411	**
	83d	Oct.	Ind.	1,187	365	**
	91st	Nov.	Oreg.	727	272	**
	99th	Dec.	Miss.	575	183	84
	10th	Nov.	Ga.	266	89	65
	11th	Nov.	Ga.	239	91	**
1943	106th	Mar.	S. C.	440	216	100
	16th	Jul.	Ark.	270	106	**
	63d	Sept.	Miss.	36	242	**

*Arranged in descending order of first three months' rate.
**Data not available.

Common respiratory disease rates for the second year of service is of the same order of magnitude for all of the units. The higher rate of the 33d Division during this period was produced by the addition of approximately 6,000 recruits to the unit in October and November of the second winter when the unit was stationed at Fort Lewis, Wash.

The sharp increase in rates shown by many of these units during November and December 1943 was due to influenza which appeared in many Army camps at that time.[2,3]

DISCUSSION

Previous studies[4,5] have demonstrated the absence of recruit epidemics in recruit populations assembled in the late summer and fall seasons. It was stated, however, that the increase in common respiratory disease during the following winter months produced a total common respiratory disease rate that was equal to the total rates of those units assembled during the winter

season with resulting recruit epidemics. The longest period of observation of these training units was 17 weeks.

Sartwell's studies at Fort Dix have demonstrated that units assembled in the fall without severe recruit epidemics did not experience excessive common respiratory disease rates during subsequent winter months. These observations were for a period of only 14 weeks.

In this study it has been possible to follow large Army units throughout the first year of service and for some units through the second year of service. These data demonstrate that large recruit populations were assembled without epidemics of common respiratory disease if assembled during the summer months, and that they did not experience epidemics during the subsequent winter season. The total common respiratory disease reported by these units during the first year following activation was approximately one fourth that of the units that experienced recruit epidemics by being assembled during the winter season in the northern states.

The divisions assembled in the winter in the northern states consistently experienced severe recruit epidemics. The units assembled in southern states during the winter months reported increased common respiratory disease at the time of assembly but did not experience the severe epidemics as did the units assembled in the northern states. The total common respiratory disease for the first year of service of these units assembled in the south was likewise of a lower order of magnitude.

It might be argued that the avoidance of epidemics during the first year by activation of units under favorable seasonal and geographic conditions merely postpones the epidemics until less favorable circumstances are encountered. Many of the units studied did move to different geographic areas after the first year of training. Despite these movements, the common respiratory disease rates after the first year were uniformly low. This is illustrated by the 95th Division, which assembled in Texas during the summer without experiencing a recruit epidemic, and reported only a normal seasonal increase in common respiratory disease upon moving to Indiantown Gap, Pa., during January and February of the second winter season following activation.

The process by which individuals entering Army organizations become more resistant to certain diseases and adjusted to military life is termed seasoning. This seasoning process includes the development of resistance to common respiratory disease, as illustrated by the experience of the divisions studied and by the experience of the total Army during the war period.[7] Troops

apparently become seasoned after four to six months. It has been demonstrated that recruit groups experience epidemics of common respiratory disease if assembled in northern states during the winter season. This study also demonstrates that units may become seasoned with respect to common respiratory disease without experiencing severe epidemics of these diseases, by assembling the recruits in the summer season or in southern states during the winter season.

The mechanism of this process of seasoning in respect to common respiratory diseases is not clear. Studies of the epidemiology of common respiratory diseases and attempts to cultivate the causative agent in different laboratories indicated that there may be several types or strains of agent.[15-19] Studies have indicated that the active immunity developed after exposure to these agents is probably strain-specific and of short duration.[19-22] Each of the strains may produce clinical illness in an individual not recently exposed to that particular strain.

The epidemiology of common respiratory disease in military organizations might be explained on the basis of these observations. The military organizations observed in this study were formed by the assembly of individuals from many geographic areas of the United States, both rural and urban. This method of formation of the units would have brought together many different strains of the causative agent of common respiratory disease, and exposed individuals in the units to many strains not previously encountered. These exposures under favorable conditions would produce infection in a large number of these individuals with a resultant immunity which would be of short duration. The immune status, however, would receive constant stimulation through continued exposure resulting from the constant close association which is so characteristic of military life. In this manner, the men would become resistant to many strains of the causative agent and become seasoned troops with respect to common respiratory disease.

The exposure to many strains of the causative agent under the conditions that exist in the assembling of units in northern states during the winter months does apparently produce infections of sufficient severity to cause large numbers of persons to be admitted to the hospitals for medical treatment. Thus recruit epidemics, as measured by hospital admission, were observed in these units.

Exposures under conditions that exist during the summer months or in units assembled in southern states during the winter season apparently produce an increased proportion of mild, subclinical attacks. Recruit epidemics, measured by hospital admissions, either did not occur or were much less extensive in

these units. In this manner the communal immunity or seasoning was developed without large numbers of hospital admissions and excessive loss of time from duty. The reason for this difference is not apparent. It suggests very strongly that there is a climatic factor which alters the host-parasite reaction. To each assembly point recruits came from various parts of the country, were processed in a similar manner, and were subjected to the same conditions of living in military life. By this process there was opportunity for viral agents of all types to be introduced. Whether the climatic factor operates on the selection of the prevalent viral strains, affects their dispersibility, or modifies the host reaction toward mildness in the warmer regions remains a problem for future investigators to solve.

SUMMARY

The experience of the United States Army during World War II has demonstrated that common respiratory diseases are a most important cause of noneffectiveness resulting from diseases. These diseases exert their greatest effect upon military populations during periods of mobilization, when large groups are being assembled from civilian life.

Common respiratory diseases reported by all Army units, and particularly by troops in the continental United States, revealed very high yearly rates and epidemic peaks during the winter months of the mobilization phase and the first two years of the war. During the last two years of the war the rates were greatly reduced and the seasonal peaks were of small magnitude. The correlation of the proportion of recruits in the Army with the common respiratory disease rates suggests that the excessive rate experienced during mobilization and the early part of the war was produced by the large number of recruits present at that time.

The Army's experience with common respiratory diseases in the various foreign theatres of operation and in the geographic subdivisions of the United States has demonstrated a definite seasonal and geographic effect upon the reported incidence of these diseases. In the northern areas the yearly incidence and the increase in incidence during the winter season were of much greater magnitude than in the southern areas.

Because epidemics of common respiratory diseases in recruit populations contribute a major part to the total Army's experience with these diseases, an inquiry into the effect of geography and season upon these diseases in large recruit populations has been conducted by the study of 19 Army divisions.

The method of activation of divisions by assembling the personnel directly from civilian life produced large recruit popula-

tions. It has been possible to follow these groups with respect to common respiratory diseases through the first year of service or longer.

This method of study has made possible the observation of very large recruit populations in various seasons of the year and in different geographic areas of the United States.

These observations reveal that geography, season, and length of military service have a distinct influence upon the incidence of common respiratory disease in Army populations, as measured by admissions to medical treatment facilities.

The highest incidence was reported during the first three or four months of service. After this period all units studied became seasoned with respect to common respiratory disease, and only minor seasonal increases in incidence were reported.

Significant differences in the magnitude of the recruit epidemics of the various divisions were observed. The units assembled in the winter months in the northern states experienced severe epidemics of common respiratory disease. Units assembled in the southern states in the same season reported increases in incidence of much lower magnitude.

Divisions assembled during the summer months recorded minor increases in incidence during the first months of service. These units did not experience epidemics during the subsequent winter season. The addition of large numbers of recruits to some of these units in the following winter season produced moderate epidemics of common respiratory disease.

Divisions assembled without recruit epidemics did not experience epidemics when unfavorable geographic and seasonal circumstances were encountered as a result of movement of the units.

These observations suggest methods by which great losses of manpower during periods of mobilization for national defense can be reduced. Under favorable geographic and seasonal conditions large recruit populations may be assembled, trained, and become seasoned without epidemics of common respiratory disease.

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First need in the reform of hospital management? That's easy! The death of all dietitians, and the resurrection of a French chef.—Martin T. Fischer

STEROID PREPARATIONS FOR TOPICAL THERAPY OF SKIN ERUPTIONS

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DURING the past three years numerous steroid preparations have been developed for use in the topical therapy of skin eruptions. Previous reports have stressed the fact that local steroid therapy is of value only in the treatment of those conditions where its effectiveness has been demonstrated.[1-8] Atopic dermatitis, contact dermatitis, seborrheic dermatitis, pruritus ani, pruritus vulvae, and erythema solare are improved by the local application of certain steroid preparations; however, relapses of these eruptions are frequent when the applications are discontinued. These compounds have proved to be of no value in the local therapy of psoriasis, chronic discoid lupus erythematosus, alopecia areata, and many other conditions. At the present time hydrocortisone free alcohol, hydrocortisone acetate, fludrocortisone acetate, and combinations of these drugs with various antibiotics are commercially available in lotions, ointments, or creams for use by the practitioner.

Although the mode of action of topically applied steroids has not been determined, Sulzberger and Witten[1] postulated the theory that the beneficial effect may be due to the local hormonal action of the steroid on the skin. Livingood and associates[9] presented evidence that fludrocortisone acetate is absorbed from the unbroken skin and may produce undesirable side effects. Witten, Shapiro, and Silber[10] were unable to demonstrate systemic absorption from the local application of hydrocortisone acetate, and recently Smith and Rosenberg[11] supported the results of these investigations. In our experience[3-8] there has been little clinical evidence of absorption from the local application either of hydrocortisone and its esters or of fludrocortisone acetate, regardless of the vehicle used.

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Read before the panel on Military Dermatology, American Academy of Dermatology and Syphilology, 7 December 1955.

The materials used in this study were supplied by Charles Pfizer & Co., Inc., Merck & Co., Inc., The Upjohn Co., Sharp and Dohme, Schering Corporation, and E. R. Squibb & Sons.

The development of new steroid compounds necessitates intensive clinical and laboratory investigation. This study has been conducted to determine the value of available steroid preparations for topical use, to assay those more recently developed, and to compare the action of the new compounds with those presently in use.

METHOD OF INVESTIGATION

Patient Selection. This study was conducted on about 4,000 patients obtained from the outpatient departments of the University Hospital and the Baltimore City Hospitals as well as on hospitalized and ambulatory private patients. Many of the patients included in this report were also included in a previous study.[8] In a number of cases follow-up was incomplete, and these cases were dropped from the study. Age and sex did not influence the study and are not considered in the results and summary tables.

Diagnoses and Evaluations. Diagnoses were confirmed by clinical consultation and laboratory studies whenever indicated. Only objective changes in the dermatoses, *i. e.*, involution of lesions, were considered in tabulating results. In most instances, if a patient did not show some improvement after one week of therapy, the preparation was considered ineffective. Every patient was evaluated by a senior investigator, and all reported results were based on blind, controlled studies.

Paired Comparison Studies. This method of study was used primarily on hospitalized cases so that the applications and observations could be more carefully controlled. The preparations were furnished to the house officers with code number labels. The key to the code was kept by the secretary and was not revealed to either house officer or senior investigator until the study was complete. Placebo ointment was applied to one part of the body and active steroid ointment or lotion to another. In the same manner, the newer steroid ointments, creams, or lotions were compared with hydrocortisone or fludrocortisone preparations already on the market.

Usage Studies. Treatment was initiated with a steroid preparation of proven value, such as 1 per cent hydrocortisone or 0.1 per cent fludrocortisone ointment, cream, or lotion. After improvement was noted, either a placebo or one of the newer compounds, in the same base and in the same size and shape of container, was substituted without the patient's or physician's knowledge, to see if improvement would continue or relapse would occur. The subjective complaints of the patient, especially in regard to pruritus, and the objective examination of the examiner were carefully noted and recorded.

In an effort to determine the optimum concentration of steroid, treatment was initiated in one series with a weak concentration

(*e. g.*, 0.5 per cent); if it proved to be ineffective, a stronger concentration was used (1.0 per cent); if this proved to be unsatisfactory, the concentration was gradually increased. In another series, treatment was initiated with a higher concentration (*e. g.*, 2.5 per cent) and gradually reduced. The lowest concentration found to be effective was termed the Minimum Effective Concentration (MEC).

Usual Treatment Method. The ointment, cream, or lotion was supplied to the patient who was instructed to apply a thin coating over the involved area twice daily. For the duration of treatment with steroid preparations, all other local and systemic therapy was discontinued and the use of soap and other cleansing compounds was forbidden. In dermatoses complicated by secondary pyogenic infection where steroid-antibiotic topical therapy was the method of choice, patients were instructed to remove crusts and other surface debris with warm water or warm saline compresses prior to the application of the medication. The application was not massaged into the site.

Vehicles Used. The various steroids and steroid-antibiotic combinations were furnished in a variety of oily base ointments, greaseless base creams, and lotion bases. The following are the formulas used:

Oily bases

No. 1 Liquid petrolatum
Wool fat
White petrolatum

No. 2 Multiwax
Cholesterol
Liquid petrolatum
White petrolatum

No. 3 Liquid petrolatum
Petrolatum

No. 4 Butyl *p*-hydroxybenzoate
Methylparaben
Multiwax
Cholesterol
Mineral oil
White petrolatum

No. 5 Plasticized hydrocarbon-gel

Greaseless bases

No. 1 Sodium lauryl sulfate
Stearyl alcohol
Cholesterol
Liquid petrolatum
Methylparaben
Propylparaben
Propylene glycol
Cetyl alcohol
Petrolatum
Water

No. 2 Zinc stearate
Polyethylene glycol
Propylene glycol
Distilled water

No. 3 Zinc stearate
Propylene glycol
Polyethylene glycol 1500
Polyethylene glycol 4000

Lotion bases

No. 1 Glycerin
Isopropanol alcohol rubbing compound

No. 3 Methylparaben
Butyl *p*-hydroxybenzoate
Propylene glycol

388837 O - 56 - 3

Lotion bases—Continued

No. 1—*Continued*
Sodium methylparahydroxybenzoate
Diglycol stearate
Petrolatum
Wax
Distilled water

No. 2 Diglycol stearate
Cetyl alcohol
Hydrous wool fat
Petrolatum
Olive Oil
Tween 40
Aseptoform
Borax powder
Sodium lauryl sulfate
Glycerin
Sulfatate B
Oil lilac flower
Menthol
Alcohol
Distilled water

No. 3—*Continued*
Polysorbate 80
Tegacid regular
Spermaceti
Deionized water

No. 4 Cetyl alcohol
Stearyl alcohol
Polysorbate 20
Span 40
Propylene glycol
Parabens
D C antifoam
Distilled water

STEROID COMPOUNDS

1-Hydrocortisone and Hydrocortisone Esters *(table 1)*. This series of compounds was used in the treatment of 1,329 patients. Hydrocortisone free alcohol and hydrocortisone acetate were used in oily base ointments, greaseless base creams, and lotion bases. In all of these vehicles concentrations of 0.5 per cent, 1 per cent, and 2.5 per cent were used. Sixty-nine different preparations of these two steroids were used in the treatment of 1,096 patients. A relatively small series of patients were treated with all of the other hydrocortisone esters except hydrocortisone hemisuccinate sodium. A total of 148 patients with various dermatoses normally responsive to local hydrocortisone therapy were treated with hydrocortisone hemisuccinate sodium ointment.

Fludrocortisone *(table 2)*. Only two compounds are included in this series. Fludrocortisone acetate in concentrations of 0.05 per cent, 0.1 per cent, and 0.25 per cent was incorporated in three oily bases and two lotion bases and applied to lesions in 1,067 patients who had dermatoses that normally will respond to local hydrocortisone therapy. 9-alpha fluorohydrocortisone tertiarybutylacetate in the concentration of 0.5 per cent in a lotion base was applied to lesions in 16 patients.

Prednisone Derivatives *(table 3)*. Four preparations were included in this portion of the study. Prednisone in a lotion base

and in an oily base was used in the treatment of 50 patients. Prednisolone succinate in an oily base was used for 24 patients. Prednisolone No. 1 was used in lotion base, two oily bases, and one greaseless base in treating 68 patients, but the percentage of primary irritation from this compound was so high that further study was discontinued. Prednisolone No. 2 is a purified compound which was used in two oily bases and one greaseless base in the treatment of 60 patients.

Allo Compounds *(table 4)*. The allo compounds are a group of synthetic steroids having complex chemical formulas. These compounds were used for clinical investigation in a limited number of patients. Use was restricted to the treatment of those dermatoses normally responsive to hydrocortisone or fludrocortisone.

Steroid-Antibiotic Combinations *(table 5)*. In this portion of the study, combinations of one or more antibiotics with hydrocortisone acetate, hydrocortisone free alcohol, fludrocortisone acetate, and prednisolone have been used in the treatment of 1,753 patients. These preparations were dispensed in several oily base ointments and several lotion bases.

Other Combinations of Steroids *(table 6)*. Combinations of hydrocortisone with Caligesic Lotion, Prantal Cream (brand of diphenmethanil methylsulfate) coal tar solution, crude coal tar, crude coal tar and Quinolor Compound Ointment, and pyridoxine hydrochloride have been used in the treatment of 226 patients.

RESULTS

Topical steroid therapy has proved to be of definite value in the management of erythema solare, atopic dermatitis, dermatitis venenata, seborrheic dermatitis, intertrigo, pruritus ani, pruritus vulvae, lichen simplex chronicus, eczematous eruption of the hands, nummular eczema, stasis dermatitis, and eczematized epidermophytosis (table 7).

One portion of this study was devoted to selection of the most suitable vehicles for treatment of the various dermatoses. The results tabulated in table 7 are based on objective findings by the examiners and not on the patients' personal preference. Two patients treated with the base containing hydrous wool fat developed evidence of hypersensitivity to that ingredient. One preparation, not mentioned in the charts, contained 1 per cent hydrocortisone in a compressed air shaving cream container, but the base proved to be a severe primary irritant and after several patients developed adverse reactions, further study with this compound was discontinued. The vehicles used in the major portion of this study were found to be satisfactory.

TABLE 1. *Hydrocortisone free alcohol and hydrocortisone esters*

Steroid	Bases used	Total number of patients	Concentration of steroid (per cent)	MEC* (per cent)	Results	
					Good	Poor
Hydrocortisone free alcohol	Oily bases 1, 2, 3, 4, 5 Greaseless bases 1, 2, 3, 4 Lotion bases 1, 2, 3, 4	747	0.5, 1.0, 2.5 0.5, 1.0, 2.5 0.5, 1.0, 2.5	1.0	701	46
Hydrocortisone acetate	Oily bases 1, 2, 3, 4, 5 Greaseless bases 1, 2, 3 Lotion bases 1, 3	349	0.5, 1.0, 2.5 0.5, 1.0, 2.5 0.5, 1.0, 2.5	1.0	328	21
Hydrocortisone t-butylacetate	Oily base 3	37	0.5	0.5	18	19
Hydrocortisone palmitate	Oily base 3	23	0.05, 1.0	1.0	20	3
Hydrocortisone hemisuccinate sodium	Oily base 3	198	0.25, 0.5	0.5	186	12
Hydrocortisone diethylamino-acetate hydrochloride	Oily base 3	19	0.5	0.5	16	3
14-hydroxy-hydrocortisone	Oily base 3	16	1.0	1.0	7	9

*Minimum effective concentration

TABLE 2. *Fludrocortisone*

Steroid	Bases used	Total number of patients	Concentration of steroid (per cent)	MEC (per cent)	Results	
					Good	Poor
Fludrocortisone acetate	Oily bases 3, 4, 5 Lotion bases 1, 4	1,067	0.05, 0.1 0.25	0.1	962	105
9-alpha-fluoro-hydrocortisone tertiary-butyl-acetate	Lotion base 1	26	0.5, 1.0	0.5	19	7

Hydrocortisone free alcohol and hydrocortisone acetate (table 1) in oily base, greaseless base, or lotion base was found to be equally effective in the treatment of responsive dermatoses (table 7). The optimum concentration in the vehicles used is 1.0 per cent. Relief of pruritus and involution of the eruption is only temporary, however, and when the applications are discontinued relapses will occur in the majority of cases. Relief of symptoms may be maintained by continued local therapy with these drugs, gradually reducing the frequency of the applications to once daily, and in some cases once every other day.

Six additional esters of hydrocortisone were studied, and of these, hydrocortisone hemisuccinate sodium and hydrocortisone diethylaminoacetate hydrochloride were effective. The MEC of those two compounds in an oily base was 0.5 per cent. By paired comparison studies and usage tests, each of these esters is as efficient as hydrocortisone or hydrocortisone acetate in a similar vehicle.

Fludrocortisone (table 2) was used in the treatment of 1,067 patients with responsive dermatoses and had an MEC of 0.1 per cent. The comparative studies between fludrocortisone and hydrocortisone indicated that the difference between the two compounds was quantitative and not qualitative. A new derivative, 9-alpha-fluorohydrocortisone tertiary-butylacetate, had an MEC of 0.5 per cent; however, the relatively low efficiency of this preparation indicated it to be of little value in dermatologic therapy.

Studies on the local efficacy of the delta-1 steroids (table 3) included observations on lotions, greaseless base creams, and oily base ointments containing prednisolone No. 1, prednisolone No. 2, and prednisolone succinate. Prednisone proved to be of little value in the treatment of normally responsive dermatoses, and in many instances (31 per cent) was productive of primary irritation on local application. The initial studies with prednisolone (prednisolone No. 1) indicated that this steroid was of value in the treatment of normally responsive dermatoses, but 25 per cent of the patients treated with this ointment developed evidence of primary irritation. Because of such reactions further studies with these compounds were discontinued. A purified compound, prednisolone No. 2, was developed for clinical investigation, and this compound was found to be efficient with an MEC of 0.5 per cent. By paired comparison and usage tests 0.5 per cent prednisolone No. 2 is as efficient as 1.0 per cent hydrocortisone or 0.1 per cent fludrocortisone. Prednisolone succinate was effective in a concentration of 0.5 per cent.

In the search for new and more effective steroid compounds for local therapy, a group of compounds with complex chemical

TABLE 3. *Prednisone derivatives*

Steroid	Bases used	Total number of patients	Concentration of steroid (per cent)	Results	
				Good	Poor
Prednisolone No. 1	Lotion base 1 Oily bases 3, 4 Greaseless base 3	68	0.5	40	28
Prednisolone No. 2	Oily bases 3, 4 Greaseless base 3	60	0.5	55	5
Prednisolone	Lotion base 1 Oily base 3	50	0.5	23	27
Prednisolone succinate	Oily base 3	24	0.5	16	8

formulas called "allo compounds" (table 4) have been developed for clinical investigation. One of these, 9-alpha fluoro-allodihydrohydrocortisone acetate, was found to be the most effective and the least irritating. 9-alpha fluoro-allodihydrocortisone tertiary-butylacetate was effective but produced a high incidence of primary irritation. No reactions of specific hypersensitivity have been produced by the members of this series which have been studied. More definite conclusions relative to the ultimate efficacy of these compounds cannot be formed on such a small series of patients. At the present time the results indicate that the members of the allo series that have been studied will be of little value for topical therapy because of the high incidence of primary irritation.

Antibiotic-steroid combinations (table 5) were valuable in the treatment of responsive dermatoses complicated by secondary pyogenic infection. The fact that the antibiotic does not inhibit the action of the steroid was demonstrated by using the various preparations on normally responsive dermatoses which were not secondarily infected. The fact that the steroid does not inhibit the action of the antibiotic was demonstrated by the prompt response of pyogenic infection to the applications. There was a low incidence of adverse reactions to any of these preparations. Gratifying results were obtained in the treatment of secondarily infected contact dermatitis of the hands of housewives.

Six different combinations of hydrocortisone and fludrocortisone with coal tar, Prantal Cream, Caligesic Lotion, and pyridoxine hydrochloride were available for study (table 6). A combination of hydrocortisone and Caligesic in a lotion base was not effective in the treatment of normally responsive dermatoses because of the high incidence of primary irritation. A combination of hydrocortisone with Prantal in a cream base proved to be par-

TABLE 4. *Allo compounds*

Steroid	Bases used	Total number of patients	Concentration of steroid (per cent)	MEC (per cent)	Results	
					Good	Poor
Allodihydrohydrocortisone acetate	Lotion base 1	13	0.5 and 1.0	0.5	8	5
9-alpha-fluoro-allodihydro-hydrocortisone acetate	Lotion base 1	31	0.5 and 1.0	0.5	22	9
9-alpha-fluoro-allodihydro-hydrocortisone tertiary-butylacetate	Lotion base 1	16	0.5 and 1.0	0.5	5	11
Allodihydrohydrocortisone tertiary-butylacetate	Lotion base 1	12	0.5 and 1.0	0.5	8	4

TABLE 5. *Antibiotic-steroid combinations*

Steroid	Antibiotic	Bases	Total number of patients	Concentration of steroid (per cent)	Results		MEC (per cent)
					Good	Poor	
Hydrocortisone free alcohol and hydrocortisone acetate	Oxytetracycline	Oily base 3	575	1.0	526	49	1.0
	Erythromycin	Oily base 3	38	1.0	33	5	1.0
	Tetracycline hydrochloride	Oily base 3	200	1.0	182	18	1.0
	Chlortetracycline hydrochloride	Oily base 3	6	1.0	5	1	1.0
	Neomycin	Oily bases 3, 4 Lotion base 3	215	1.0	200	15	1.0
	Neomycin-bacitracin	Oily bases 3, 4 Lotion base 3	70	1.0	62	8	1.0
	Neomycin-bacitracin-polymyxin B sulfate	Oily base 4	62	1.0	54	8	1.0
	Oxamycin	Oily base 3	50	1.0	45	5	1.0
	Oxamycin-MK63	Oily base 3	39	1.0	34	5	1.0

TABLE 5. *Antibiotic-steroid combinations*—Continued

Steroid	Antibiotic	Bases	Total number of patients	Concentration of steroid (per cent)	Results		MEC (per cent)
					Good	Poor	
Fludrocortisone acetate	Neomycin	Oily base 4 Lotion base 1	67	0.1 and 0.25	61	6	0.1
	Neomycin-tyrothricin	Oily base 3 Lotion base 1	32	0.1 and 0.25	30	2	0.1
	Neomycin-bacitracin	Oily base 3 Lotion base 1	71	0.1 and 0.25	66	5	0.1
	Neomycin-gramicidin	Oily base 5 Lotion base 4	250	0.1	222	28	0.1
	Nystatin	Oily base 5	20	0.1	18	2	0.1
Prednisolone	Neomycin-bacitracin	Lotion base 1 Oily base 3	28	0.5	16	12	0.5
	Neomycin	Lotion base 1 Oily bases 3, 4	30	0.5	21	9	0.5

ticularly effective in the treatment of intertrigo. In the treatment of chronic lichenoid dermatitis, combinations of hydrocortisone with coal tar solution and fludrocortisone with crude coal tar and Quinolor Compound Ointment were effective in producing relief of symptoms. A combination of hydrocortisone with pyridoxine was used in the treatment of seborrheic dermatitis, but comparative studies and usage tests indicate that this preparation is not superior to hydrocortisone ointment or lotion. It is evident that the addition of pyridoxine hydrochloride to a preparation containing a steroid does not add to the efficacy of the steroid ointment.

TABLE 6. *Other steroid combinations*

Steroid		Total number of patients	Concentration of steroid (per cent)	Results	
				Good	Poor
Hydrocortisone	Caligesic Lotion	22	0.5	13	9
Hydrocortisone	Prantal Cream	68	.25	60	8
Hydrocortisone	Coal tar solution	22	1.0	18	4
Hydrocortisone	Crude coal tar	10	1.0	6	4
Fludrocortisone	Crude coal tar and Quinolor Compound Ointment	60	0.1	53	7
Hydrocortisone	Pyridoxine hydrochloride	44	1.0	31	13

In the course of this investigation, topical steroid therapy proved to be of *no* value in the treatment of:

Psoriasis
Lichen planus
Chronic discoid lupus erythematosus
Morphea
Keratosis follicularis
Pityriasis rosea
Alopecia areata
Epitheliomata
Acne vulgaris
Cutaneous fungus infections
Pustular bacterid
Herpes simplex
Verrucae

COMMENT

The local preparations of hydrocortisone free alcohol, hydrocortisone acetate, fludrocortisone acetate, steroid-antibiotic combinations, and some of the other combinations of hydrocortisone were found to be valuable additions to the armamentarium of the physician provided that he takes into consideration the fact that these preparations seldom produce more than temporary relief from symptoms. In many instances, the necessity for systemic steriod therapy has been obviated by the proper use of the topical preparations. It has been possible to discontinue systemic ad-

TABLE 7. *Dermatoses definitely benefitted by topical steroid therapy*

Dermatoses	Number of patients		Most satisfactory vehicle
	Improved	Unimproved	
Erythema solare	60	0	Lotion, greaseless base cream
Atopic dermatitis	735	95	Lotion, oily base, greaseless base cream
Atopic dermatitis (complicated by pyogenic infection)	300	30	Antibiotic-steroid preparation in lotion or oily base
Dermatitis venenata (all causes)	450	70	Lotion, greaseless base cream
Dermatitis venenata (complicated by pyogenic infection)	301	20	Antibiotic-steroid preparation in lotion or oily base
Seborrheic dermatitis	128	23	Oily base, or greaseless base
Seborrheic dermatitis (complicated by pyogenic infection)	92	9	Antibiotic-steroid preparation in lotion or oily base
Intertrigo	91	4	Lotion, greaseless base
Pruritus ani	242	31	Oily base, lotion
Pruritus vulvae	135	22	Oily base, lotion
Lichen simplex chronicus (neurodermatitis)	175	32	Oily base, greaseless base
Eczematous eruption of hands	330	41	Oily base, antibiotic-steroid combination in oily base
Nummular eczema	37	3	Oily base
Stasis dermatitis	123	23	Oily base, greaseless base
Eczematized epidermophytosis	22	4	Oily base

ministration of steroids in some individuals by the use of the ointments, creams, or lotions containing hydrocortisone, hydrocortisone acetate, and fludrocortisone. Dramatic relief has been afforded many patients with pruritus ani, pruritus vulvae, localized neurodermatitis, atopic dermatitis, and contact dermatitis.

CONCLUSIONS

Hydrocortisone free alcohol and hydrocortisone acetate are effective in the treatment of responsive dermatoses when applied locally in concentrations of 1 per cent or greater and in suitable vehicles. Hydrocortisone hemisuccinate sodium and hydrocortisone diethylaminoacetate hydrochloride in concentrations of 0.5 per cent are both effective in the treatment of responsive dermatoses. By paired comparison studies and usage tests, a concentration of 0.5 per cent of these two compounds is as effective as 1 per cent hydrocortisone free alcohol or hydrocortisone acetate.

Fludrocortisone is effective in the treatment of responsive dermatoses when applied locally in concentrations of 0.1 per cent or greater and in suitable vehicles. The difference in action between hydrocortisone, hydrocortisone esters, and fludrocortisone is quantitative, not qualitative.

Prednisone is relatively ineffective on topical application. Initial studies on prednisolone indicated this compound to be therapeutically effective but productive of primary irritation in 25 per cent of the patients treated. Recent studies with purified prednisolone indicate that this steroid is consistently beneficial in the treatment of responsive dermatoses without the production of primary irritation.

The allo compounds included in this study have proved to be of little value because of the high incidence of primary irritation produced by the topical applications. The addition of antibiotics to ointments, creams, or lotions containing topically active steroids does not alter the effect of the steroid and has the additional therapeutic advantage of eradicating any secondary pyogenic infection that may be present. These steroid-antibiotic combinations proved to be of value in the treatment of responsive dermatoses complicated by secondary pyogenic infection.

Combinations of hydrocortisone with Caligesic Lotion proved to be of little value because of primary irritation produced. Hydrocortisone added to Prantal Cream was of value in the treatment of uncomplicated intertrigo. Combinations of fludrocortisone and hydrocortisone with coal tar are valuable in the treatment of lichenified dermatitis.

Topical steroid therapy proved to be of no value in the following conditions: psoriasis, lichen planus, chronic discoid lupus

erythematosus, morphea, keratosis follicularis, pityriasis rosea, alopecia areata, epitheliomata, acne vulgaris, cutaneous fungus infections, pustular bacterid, herpes simplex, and verrucae.

In self-limited inflammatory conditions, the topical application of the steroid offers immediate relief to the patient and may prevent such a dermatosis from becoming chronic. In responsive chronic dermatoses, topical steroid therapy may offer a maximum relief to the patient, but in these eruptions it does not produce permanent involution of lesions. In many instances, topical steroid therapy may obviate the necessity for systemic steroid therapy.

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"Within 25 years there will be, in the adult population, three special persons for every able-bodied worker. There will be: one physically handicapped, one chronically ill, one over 65 years old.

"Nutrition can help every one of these groups to better and more useful lives."

—L. B. PETT, M. D.
in *Canadian Services Medical Journal*, p. 200, Mar. 1956

RECENT PROGRESS IN EXPERIMENTAL ELECTROCARDIOGRAPHY

J. A. ABILDSKOV, *Captain, MC, USAR*

DURING the past several years the technics employed in clinical electrocardiography have undergone relatively little change. The 1943 recommendations of the American Heart Association's committees on standardization of electrocardiographic nomenclature and precordial leads have been widely adopted, and both the range of usefulness and the limitations of the electrocardiogram as currently used have been well defined. In contrast to the static state of clinical electrocardiography, important experimental observations have been made during this period. Because the new information obtained will undoubtedly influence clinical use of the electrocardiogram, it seems desirable to summarize and view in perspective certain of the recent studies.

HISTORICAL PERSPECTIVE

Although many workers have made significant contributions to electrocardiography, two are especially noteworthy both for the merit of their work and for their influence on the approach taken by others in this field. The early history of electrocardiography is dominated by the work of Sir Thomas Lewis. In a surprisingly short time use was made of his observations to form an important part of the clinical evaluation of patients. The electrocardiographic characteristics of the major arrhythmias were identified by Lewis, making possible the important therapeutic and prognostic implications of precisely identified mechanismal disturbances. Dr. Frank N. Wilson played a similar role in the later history of electrocardiography. His contributions may be summarized as the application of mathematics and basic principles of physics to the study of the electrocardiogram, and resulted in bringing order to a field which would otherwise consist of a bewildering collection of empiric observations. Wilson's work has also had the important effect of encouraging further work in which mathematics and the fundamental principles of physics and other disciplines are employed. This approach, together with improving instrumentation and experimental technics, has resulted in significant progress in several areas related to electrocardiography.

From William Beaumont Army Hospital, Fort Bliss, Tex.

ORIGIN OF BIOELECTRIC PHENOMENA

Considerable clinical usefulness of the electrocardiogram was achieved in spite of only meager knowledge of the processes which give rise to electric phenomena in the heart. It seems certain that identification of these processes will result in an even greater range of usefulness. The origin of bioelectric potentials is by no means completely understood at this time, but radioactive isotope technics and modern physical and enzyme chemistry have provided important information in this area. Studies of nerve tissue employing radioactive isotopes have demonstrated a movement of sodium ions into the interior of cells during the rising phase of action potentials and a movement of potassium out of the cell during the falling phase.[1] These ionic movements must be set in motion by chemical processes, and there is evidence that acetylcholine is the agent responsible for altered ion permeability.[2] A working concept of the origin of bioelectric potentials is that acetylcholine exists in a bound inactive form in the resting state. Excitation of the cell by any means results in a release of acetylcholine which acts on a specific receptor and results in permeability changes which are followed by the ionic movements described. The acetylcholine is rapidly inactivated by the enzyme acetylcholinesterase.

SEQUENCE OF ELECTRICAL EVENTS IN THE HEART

The structure of clinical electrocardiography has been built on limited knowledge of the sequence in which electrical events involve various portions of the heart. The general path of activation in the atria was relatively easily defined because these structures are thin and for most purposes the spread of electrical events through them may be considered as a surface phenomenon. In the ventricles, however, the sequence of activation on the surface cannot be presumed to indicate the same as that in the underlying muscle. Several recent studies have provided important information on the detailed sequence of activation throughout the thickness of the ventricles.

Scher and Durrer and their co-workers[3-10] used similar technics in investigations of the order of ventricular activation in dogs and other animals. Both used multiple small electrodes on needles inserted in the ventricular muscle at a variety of sites and recorded electrical events with cathode-ray oscilloscopes, employing technics which permitted time relations in the range of milliseconds to be recognized. Both investigators confirmed earlier studies indicating that the general sequence of activation of the left ventricular wall proceeds from apical toward basal regions. Scher's studies, which included study of the intraventricular septum, also confirmed the finding that the left side of the septum was the earliest portion of the ventricular muscle to

be activated. His results, however, indicate activation beginning in the midportion of the left septal endocardium, whereas the classic study of Lewis and Rothschild[11] indicated that this occurred high on the septal wall, and later Sodi-Pallares and associates[12] and Burchell, Essex, and Pruitt[13] found evidence that the apex of the septum was excited before the base. Results obtained by Scher and Durrer and their associates differed with regard to the manner of excitation in the subendocardial regions. Both found a very rapid activation of the endocardial surface of the ventricular wall. Studies of Scher and co-workers, however, indicated a spread of activation through the remainder of the ventricular wall in an endocardial to epicardial fashion. Results of studies made by Durrer and associates seem to indicate myocardial extensions of the Purkinje system so that activation in the inner layers may be progressing toward the endocardial as well as epicardial surfaces. In this respect findings of Durrer and associates are similar to those obtained by Prinzmetal and coworkers.[14] Despite the areas of conflict in the studies of Scher and associates and those of Durrer and his co-workers, the application of the technics employed by these investigators represents an important step toward more complete understanding of the electrocardiogram. When the detailed order of activation has been defined it will be possible to postulate the electrocardiographic effects of lesions in any location and it seems probable that the range of clinical usefulness of the electrocardiogram will be extended.

METHODS OF ELECTRODE PLACEMENT

In clinical electrocardiography, electrodes on the limbs and at selected sites on the precordium are employed. The limb electrodes are assumed to define an equilateral triangle with the heart acting as a point source of dipole current while the precordial electrodes are considered to act in a fashion similar to electrodes placed directly on the surface of the heart. Both of these assumptions are known to be erroneous, but detailed quantitative investigations of the errors have only recently been carried out. Still more important, methods of reducing these errors are being developed. These methods are likely to be used clinically in the near future, and together with the increased knowledge of cellular events responsible for bioelectric potentials and of the sequence of spread of electrical phenomena in the heart as well as improved methods of registering these phenomena, the range and accuracy of clinical electrocardiography seems certain to be extended.

Important investigations of the validity of assuming the heart to be a point source of dipole current have been carried out by Schmitt and associates.[15-17] These workers used the property of mirror images of the potential from a dipole source at opposite

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points on a line through the source. The degree to which canceling mirror patterns of electrocardiographic leads could be found on the surface of the body of both normal and abnormal subjects was studied. Their results indicated that approximately 90 per cent of the electrical potential appearing at any point on the body may be ascribed to a dipole-like source at the heart. Similar studies by Frank[18] indicated that the QRS complex at all points on the body of normal subjects can be considered to arise from a dipole source with an average accuracy of 5 per cent. Detailed studies of a single subject by Frank[19] also support the validity of the dipole concept. In these studies, potentials at the surface of a model of the subject's torso produced by an actual dipole source were compared with QRS complexes recorded from the subject. Both amplitude and wave form were found to correspond to an accuracy of ± 15 per cent.

These studies have the important connotation of questioning the validity of the "semidirect" lead concept. The results tend to indicate that most of the information which can be obtained from the surface of the body concerning electrical events in the heart is present in leads recorded from distant or remote electrode sites. This is especially important when considered in association with the advances in recording methods. It would mean that a vectorcardiogram recorded from electrode sites yielding horizontal, vertical, and anteroposterior components of electrical activity in the heart would contain not only most of the information available in the limb and precordial leads in current use but most of the information about electrical activity in the heart that it is possible to obtain from electrodes anywhere on the surface of the body. A great deal of clinical experience will of course be necessary to confirm this thesis on a practical level and to define the form in which the clinically important information presents itself.

The limb leads currently employed in clinical electrocardiography reflect the heart's electrical activity in only one plane. To some extent the precordial leads reflect electrical activity in other planes but it is still difficult to visualize this activity as it occurs in space. The vectorcardiogram permits such a view of electrical activity, and increasing interest in records of this type has focused attention on methods of electrode placement which will permit the least distorted view of this activity. Many systems of electrode placement have been suggested. Most of these are simple geometric arrangements such as the tetrahedron, parallelepiped, or cube. Frank's[20] studies have indicated that among these electrode arrangements, the equilateral tetrahedron yields the most valid indication of the heart's electrical activity. All of these electrode systems, however, are known to result in significant errors, and it is highly desirable that more adequate

systems be devised. One approach to this problem is represented by the work of McFee and Johnston.[21-23] These workers employed plaster models of selected planes of the body, through which water was made to flow in sheets. The models were so arranged that resistance to the flow of fluid in them was analogous to electrical resistance in the body and the flow lines of the water were made visible by small crystals of a soluble dye. With these fluid mappers the pattern of flow lines between various electrode combinations could be visualized and it was possible to select combinations of electrodes in which the undesirable features of the flow lines tended to cancel one another. From these studies electrode combinations to yield horizontal and anteroposterior components of the heart's electrical activity were suggested. Electrodes on the right and left arms are paired with electrodes on the right and left sides of the thorax to yield the horizontal component, and multiple electrodes in the front and back of the chest are employed to obtain the anteroposterior component.

Extensive studies by Frank[24] and by Schmitt and Simonson[25] employing torso models have resulted in other suggested methods of electrode placement. At the present time it seems that additional experimental work is still necessary to define the optimum electrode combinations, and from the progress to date this should soon be achieved. Application of this improved electrode system of the future is certain to provide more accurate electrocardiograms and vectorcardiograms and is very likely to improve the clinical usefulness of these records.

PRESENTATION OF ELECTROCARDIOGRAPHIC INFORMATION

The conventional electrocardiogram is a Cartesian co-ordinate graph in which voltage is plotted against time. The quantities represented are scalars, having the properties of magnitude and sign. Many methods employed in interpretation of electrocardiograms, such as the estimation of mean electrical axis, are examples of vector analysis. In the application of such methods to the electrocardiogram, vector quantities which have the property of specific direction as well as that of magnitude are derived from the scalars. It has been obvious for many years that actual recording of electrical events from the heart as vectors might have certain advantages over recording scalar quantities. Electronic amplifiers and cathode-ray oscilloscopes have made it possible to obtain such records, and during the past few years they have been subjected to extensive study. Even though these studies have been handicapped by the inadequacies of the electrode systems employed, there are many indications that vectorcardiograms will eventually furnish clinically important information in addition to that provided by the conventional electrocardiogram.

Despite certain potential advantages, the vectorcardiogram recorded from cathode-ray oscilloscopes has a major disadvantage in the difficulty of representing time in such a record. It seems possible to retain the advantages of a vector presentation and yet have time adequately represented by presenting magnitude and orientation of spatial vector forces as separate traces on a Cartesian co-ordinate graph. Such a method of presentation was suggested by McFee,[26] who reported construction of a computer allowing registration of such traces from one plane of the body. More recently Sayers, Silberberg, and Durie[27] reported observations on curves of spatial magnitude only. Other observations on curves of both spatial magnitude and orientation have indicated that the information known to be clinically important in the electrocardiogram and the vectorcardiogram is present in easily accessible form in such records.[28] These observations have also indicated that certain variations in spatial magnitude of recorded electrical events from the heart are made apparent by this method of presentation even when they are not easily evident in the electrocardiogram or vectorcardiogram recorded from cathode-ray oscilloscope. Study of these tracings in which vectorcardiographic data is presented on a linear time scale is just beginning, but it offers considerable promise for more precise study of electrical phenomena arising from the heart than has been possible previously.

Considerable progress has been made in obtaining a quantitative expression of the relation between activation and recovery, and this relation is the basis of the concept of the ventricular gradient. Electronic devices for the automatic determination of this quantity have been devised.[29-31] Assessment of the final value of this quantity in clinical practice will require extensive correlation with other clinical, laboratory, and pathologic data.

SUMMARY

While clinical electrocardiography has changed very little for several years, there has been considerable progress in this field on the experimental level. This progress has ranged from new information concerning the cellular processes which give rise to electric potential, to improvements in the methods of recording, and manner of presenting records of electrical activity in the heart. There have also been refined and detailed studies of the sequence of spread of electrical processes through the heart and important advances in methods of electrode placement to extract the most valid information about these processes from the surface of the body. Progress in all of these areas is pertinent to clinical electrocardiography and seems certain to extend the range and accuracy of this method of diagnosis.

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SURGERY AND THE CORONARY

"It now appears that, if a patient has survived his acute coronary heart episode and has made the transition into the chronic stage, his outlook for a long and productive life is good enough to warrant almost any operation that would otherwise be justifiable. It also appears that under such circumstances the risk of surgery is negligible insofar as cardiac deaths are concerned. This reassuring fact should make patients with coronary heart disease much less reluctant to submit to recommended surgery and much less frightened when they do."

—BENJAMIN ETSTEN, M. D., and
SAMUEL PROGER, M. D.
in *Journal of American Medical Association*
p. 848, Oct. 29, 1955

CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEOESOPHAGEAL FISTULA

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ROSCOE E. MASON, *Major, MC, USA*
GEORGE L. BEATTY, *Colonel, MC, USA*

THE NECESSITY for prompt surgical correction of congenital atresia of the esophagus with tracheoesophageal fistula is widely recognized. The diagnostic criteria of excessive salivation, regurgitation, with or without cyanosis, and the demonstration of a blind esophageal pouch by catheter and fluoroscopic study have been well documented. The surgical procedures for the correction of this anomaly have been standardized. We here record the experience at this hospital to emphasize more fully some of the practical problems which arise in the management of these infants.

This anomaly was described by Gibson in 1703.[1,2] It was not until 1939 that surgical treatment was successfully accomplished. This was done by closure of the fistula and exteriorization of the proximal esophageal pouch by Leven[3] and by Ladd.[4] Haight and Towsley[5] performed the first successful primary esophageal anastomosis for this condition which was reported in 1943. Since that time the advances in surgery of the newborn infant have enabled many of the large centers to report from 60 to 80 per cent survival after operation.

Any discussion of this subject would be incomplete without reference to classification as indicated graphically (fig. 1) as adapted from Ladd[2,4] and Vogt.[6] Congenital anomalies are more common among premature infants, and in many instances, are multiple. The incidence of tracheoesophageal fistula is variously recorded as between 1 in 800 and 1 in 2,500 births. At this hospital there have been five cases over a three-year period, extending from February 1952 to February 1955. During this time there were a total of 9,320 births, including 106 stillbirths. This is an incidence of 1 case of esophageal atresia with tracheoesophageal fistula per 1,864 births. This anomaly occurred in one premature stillbirth and was discovered at autopsy. The remaining four cases were in full-term infants in whom the diagnosis was made and an operation performed. In three instances the fistula entered

From Tripler Army Hospital, Honolulu, T. H.

the trachea at the level of the carina, while in the fourth the fistulous opening was 1.5 cm above the carina, in the posterior, or membranous portion, of the trachea.

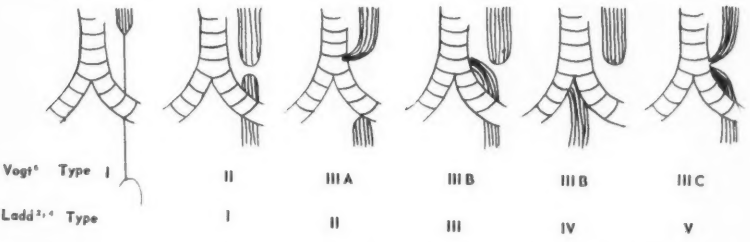

Figure 1. Schematic representation of esophageal atresia. The Vogt type I, congenital absence of the esophagus, is rare. The Vogt type II (Ladd type I), blind upper and lower esophageal segments, is rare. The Vogt type III (Ladd types II, III, IV, V) represents the various possible tracheoesophageal fistulas associated with atresia. The most common anomaly is the Vogt type III B (Ladd types III and IV).

The presence of esophageal atresia was suspected within 30 hours of birth in three instances when the nursery staff noted regurgitation, excessive salivation, and intermittent cyanosis.

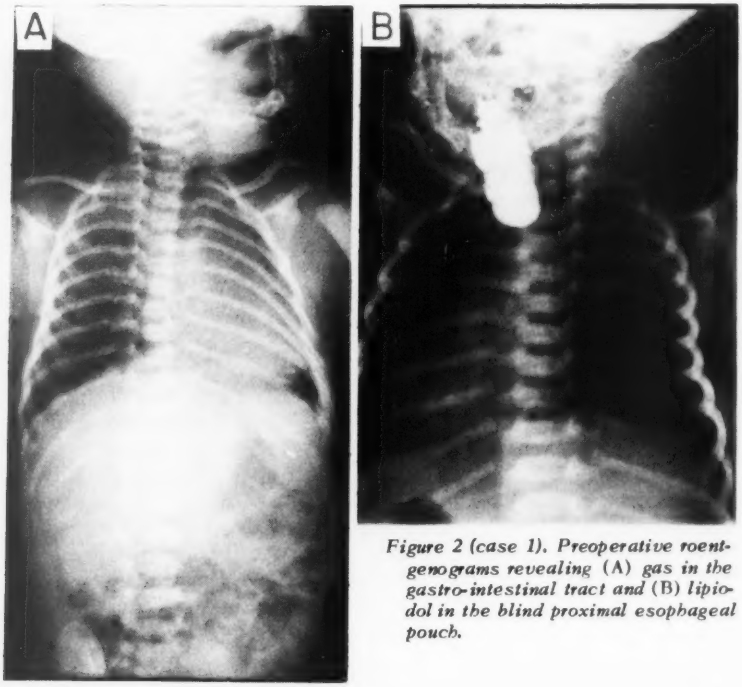

Figure 2 (case 1). Preoperative roentgenograms revealing (A) gas in the gastro-intestinal tract and (B) lipiodol in the blind proximal esophageal pouch.

In the fourth case the presence of esophageal atresia was not suspected and diagnosed until the third day of life. In all instances the diagnosis was confirmed by radiologic demonstration of the blind proximal esophageal pouch as well as gas in the stomach and small intestine. Supportive therapy was administered over a period of several hours and the operation was performed as an emergency procedure.

REPORT OF CASES

Case 1. A 7 lb 9 oz baby girl was born on 8 February 1952. The pregnancy and delivery were uncomplicated. The only abnormality noted at birth was a small hemangioma of the right foot. During the first 24 hours of life an excessive accumulation of mucus was noted, and regurgitation occurred. The diagnosis of esophageal atresia with tracheoesophageal fistula was suspected and verified by a lipiodol esophagram (fig. 2). The infant was subsequently taken to the operating room where a vein was cannulated and endotracheal anesthesia administered. A transpleural approach through the bed of the resected fourth rib was accomplished. The fistula was identified at the carina and ligated. A primary anastomosis of the esophagus was made with moderate tension on the suture line. A nasogastric catheter was inserted to splint the anastomosis. The chest wall was closed with drainage through an intercostal catheter. The patient was given penicillin and streptomycin sulfate postoperatively. On the second postoperative day because it was discovered that the nasogastric tube had been accidentally displaced above the anastomosis it was removed. Water was administered orally by dropper. A hydrothorax developed shortly thereafter. At this time leakage at the site of anastomosis was demonstrated roentgenographically by the administration of a small amount of lipiodol. On the third postoperative day the infant developed respiratory distress with cyanosis. Suction applied to the thoracotomy tube resulted in the aspiration of small amounts of fluid. A catheter was reinserted into the esophagus and Wangensteen suction was applied. On the sixth day after surgery, cyanosis became constant and the baby died. At autopsy dehiscence of the anastomosis with rupture of a mediastinal abscess into the right pleural space was demonstrated.

Comment. At operation the anastomosis was noted to be under moderate tension. A gastrostomy was not performed in this instance. This, in retrospect, might have modified the postoperative course. If a gastrostomy had been done and feedings administered by this route postoperatively, a better result might have been expected. Death was the result of mediastinitis secondary to separation of the suture line.

Case 2. A 6 lb 6 oz baby girl was born on 10 August 1953. The pregnancy and delivery were uncomplicated. During the first 30 hours of life the infant appeared to have an excessive amount of mucus. An attempt to pass a gavage tube was unsuccessful. The diagnosis of

esophageal atresia with tracheoesophageal fistula was suspected and was confirmed by lipiodol esophagram (fig. 3). The chest roentgenogram at this time revealed atelectasis of the right upper lobe. After cannulation of a vein the infant was taken to the operating room. Under endotracheal anesthesia a transpleural approach through the bed of the resected fifth rib was made. The fistula was identified at the carina and closed. A satisfactory two-layer anastomosis of the esophagus with minimal tension was accomplished. On two occasions during the procedure cardiac massage was required to reinforce the feeble contractions. A nasogastric tube and intercostal catheter were left in place. A gastrostomy was performed. The patient was maintained on intravenous fluid therapy for 72 hours, at which time gastrostomy feeding was instituted and the nasogastric tube removed. The feedings were gradually increased in amount. On the ninth postoperative day oral feedings were begun and shortly thereafter the gastrostomy tube was removed. Fifteen days postoperatively a lipiodol esophagram appeared normal (fig. 4). Penicillin and streptomycin sulfate were administered postoperatively. A month after surgical intervention the infant was discharged from the hospital and subsequently lost from follow-up when the parents were transferred from this community.

Comment. In this instance, in spite of known preoperative atelectasis and a precarious situation at operation requiring cardiac massage, a good anastomosis with minimal tension was

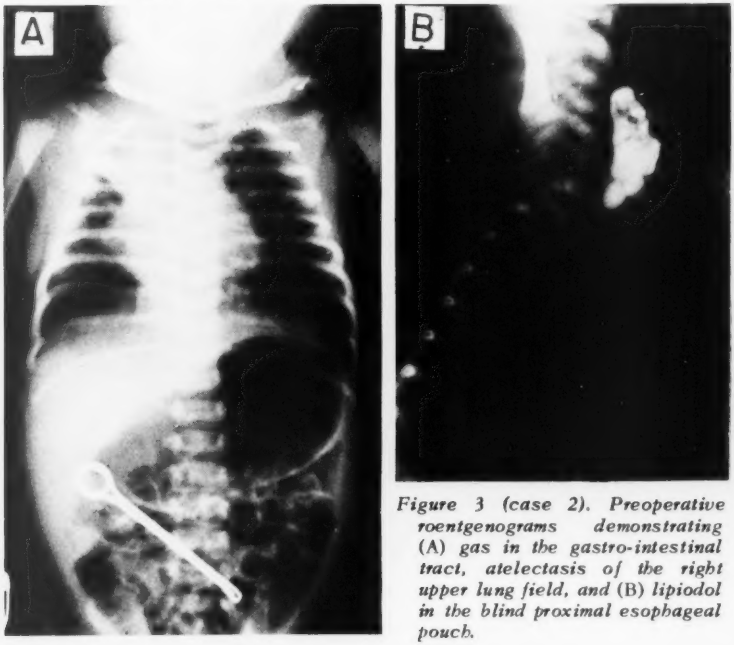

Figure 3 (case 2). Preoperative roentgenograms demonstrating (A) *gas in the gastro-intestinal tract, atelectasis of the right upper lung field, and* (B) *lipiodol in the blind proximal esophageal pouch.*

obtained. Gastrostomy feedings were administered early in the postoperative period without danger to the esophageal repair and the baby survived.

Case 3. This 6 lb 15 oz baby girl was born on 6 October 1953. The pregnancy and delivery were uncomplicated. Within eight hours the infant was noted by the nursery staff to be producing an abnormal amount of mucus. At this time several episodes of cyanosis occurred. An attempt to pass a gavage tube was unsuccessful, and a diagnosis of esophageal atresia with tracheoesophageal fistula was confirmed by lipiodol esophagram (fig. 5). The patient was operated on within the first 24 hours of life. A vein was cannulated and endotracheal anesthesia administered. A transpleural approach through the bed of the resected fourth rib was performed. The fistulous tract was identified entering the carina; this was divided, and the trachea repaired. During the dissection of the blind proximal pouch of esophagus from the trachea, the latter was inadvertently opened a distance of 1 cm in the posterior membranous portion. This opening was immediately repaired. An end-to-end, two-layer anastomosis of the esophagus over a catheter was obtained. There was moderate tension of the suture line. The nasogastric tube was left in place and an intercostal catheter inserted during closure of the chest. Postoperatively the baby was cyanotic when out of the oxygen tent and appeared more irritable than expected. A roentgenogram of the chest at this time revealed haziness of the left upper lung field. Twenty-four hours postoperatively the infant began having irregular, grunting respiration. Respiration gradually became rapid and irregular over the next several days, and the baby died of respiratory insufficiency on the third postoperative day. Autopsy permission was not obtained.

Figure 4 (case 2). Postoperative lipiodol esophagram demonstrating an intact esophagus.

Comment. This case points out the seriousness of pulmonary complications. These should be considered as a part of the pathologic condition in

all of these infants. This was a difficult operative procedure during which the trachea was opened. The addition of a tracheotomy might have provided a better chance for survival.

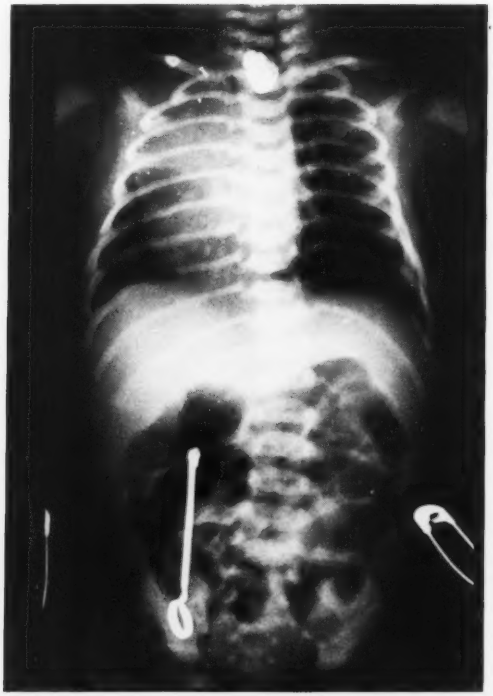

Figure 5 (case 3). Preoperative roentgenogram revealing the presence of gastro-intestinal gas, as well as the proximal esophageal pouch outlined with lipiodol.

Case 4. This 6 lb 2 oz baby boy was born on 27 January 1955. The pregnancy and delivery were uncomplicated. The infant was allowed to remain at the mother's bedside in a bassinet, in keeping with the "rooming-in" policy of the obstetrical service. The baby was, therefore, cared for by the mother who was not aware of the significance of persistent regurgitation. When the infant was two days of age the nursery staff became aware of the situation when an episode of cyanosis was observed. The diagnosis of esophageal atresia with tracheoesophageal fistula was confirmed on the third day of life by lipiodol esophagram (fig. 6). A vein was cannulated and endotracheal anesthesia administered. The thoracic cavity was entered through the bed of the resected fourth rib. The distal esophagus was identified and the fistulous tract found entering 1.5 cm above the carina in the posterior mem-

branous portion of the trachea. The fistula was closed by suture and a two-layer esophageal anastomosis with minimal tension accomplished. A gastrostomy and tracheotomy were performed at the conclusion of the procedure. A nasogastric catheter was left in place as well as an intercostal tube. The nasogastric catheter, intercostal tube, and tracheotomy tube were removed 24 hours postoperatively. The infant was maintained on fluids given intravenously for 48 hours. Penicillin and streptomycin sulfate were administered. On the third postoperative day gastrostomy drip feeding of glucose water was begun. The amount and strength of feeding was gradually increased until several ounces of formula for newborn infants were given every three hours. On the sixth postoperative day, oral feedings in small amounts were started. The patient continued to do well throughout the remainder of hospitalization. A follow-up esophagram revealed good function (fig. 7), and the baby was discharged from the hospital. At the age of six weeks the infant was seen in the outpatient clinic and was doing well.

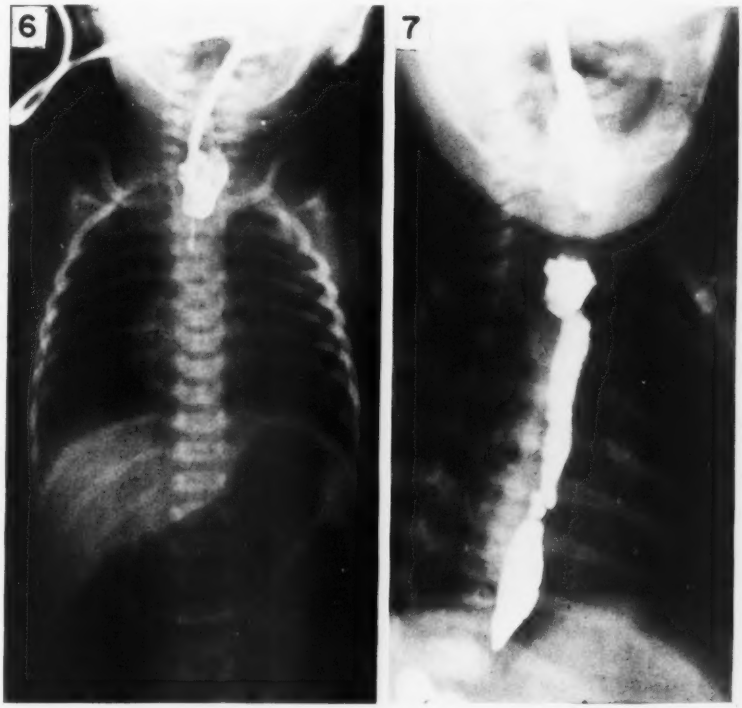

Figure 6 (case 4). Preoperative roentgenogram with lipiodol in the proximal esophageal pouch. Note the gas in the stomach and intestines. Figure 7 (case 4). Postoperative lipiodol esophagram. The arrow points to the site of anastomosis.

Comment. The location of the fistula high in the trachea above the carina was fortunate in that a good anastomosis with minimal tension was possible. Early postoperative pulmonary complications were anticipated and prevented by tracheotomy which was added to the operative procedure. The value of tracheotomy in this instance may be questioned; however, it did not increase the operative procedure appreciably and certainly did not increase the risk.

DISCUSSION

The diagnosis and surgical treatment of esophageal atresia with tracheoesophageal fistula have been adequately described in the literature. [1-5, 7-11] The postoperative management of these infants assumes equal importance if one is to obtain consistently satisfactory results. The administration of fluids and blood through a vein requires a preoperative "cut down" in the newborn infant. This provides for continuous administration of fluid during the critical 48- to 72-hour postoperative period. However, meticulous and constant observation of the flow rate must by maintained to prevent pulmonary edema. We accomplish this by making a detailed chart of the rate of flow in milliliters of fluid per hour to be administered, based on the 24-hour requirement. A blank column is provided on the chart which is filled in at hourly intervals by the nursing staff recording the actual volume of fluid given. Fluids are administered from a modified 200-cc graduated buret so that an accurate record can be maintained and the danger of overhydration minimized. An intercostal catheter must be left in the thorax and attached to a water-seal drainage bottle for at least 24 hours or longer to ensure expansion of the lung and a dry pleural cavity. Precautions commonly utilized in thoracic surgery, namely, endotracheal anesthesia and antibiotic therapy, have permitted the more satisfactory transpleural approach. It is tempting to rely on the nasogastric catheter placed at operation to prevent regurgitation of gastric secretions to the site of anastomosis in the immediate postoperative period, and to institute tube feeding within 48 to 72 hours. In our limited experience it would seem that a gastrostomy is more satisfactory. Certainly, for those who are only occasionally called on to perform such an operation, a gastrostomy is indicated. Continuous drip feeding may be administered by this route within 24 to 48 hours after the operation.

The dehiscence of the esophageal anastomosis in the first baby treated resulted in fatality. A gastrostomy performed as a part of the initial operation would have provided a satisfactory feeding route which was not otherwise available. Disruption of the anastomosis must be anticipated and recognized as a possible complication. Mediastinal drainage in the first case at the time of disruption would have improved the possibility of survival. It must be assumed that all of these infants have aspira-

tion pneumonia and atelectasis. The importance of obtaining and maintaining expansion of the lungs cannot be overemphasized. It must also be assumed that the older the baby is before diagnosis, the more serious the pulmonary complications are likely to be. For this reason we believe that tracheotomy should be considered in those patients in whom the diagnosis or surgical intervention is delayed. This concept is not presented in the surgical literature as a part of the treatment for congenital esophageal atresia with tracheoesophageal fistula. We believe that this is of value in selected patients as it provides adequate access to the tracheobronchial tree for aspiration and improves pulmonary function by decreasing the dead space volume. The provision of a warm, moist atmosphere with a high oxygen content is an important adjunct in management during the critical immediate postoperative period. Antibiotic therapy has certainly been of value in minimizing secondary infection, but will not substitute for good surgical technic.

SUMMARY

We have presented our brief experience with four cases of surgically treated infants having esophageal atresia with tracheoesophageal fistula. There were two fatalities. It has been pointed out that the limiting factor in the operative decision to perform esophageal anastomosis is the amount of tension on the suture line. The first fatality resulted from the disruption of the anastomosis. The addition of a gastrostomy to the operative procedure allows safe early feeding. Emphasis has been placed on the postoperative management of these patients. All of these infants must be treated for aspiration pneumonia. Tracheotomy in those babies in whom the diagnosis or treatment has been delayed may be indicated.

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INFANT FEEDING

"There is another tendency in modern infant feeding which needs to be brought under critical scrutiny. This is the practice of beginning solid food supplements at increasingly early ages. Early in the 1920's, solid food additions were made rather cautiously during the latter months of the first year. Marriott, in the second edition of his excellent book on infant nutrition published in 1935, states that the appropriate age to begin solid food additions is six months. Today, many infants are having their milk diets supplemented by solid foods as early as the second and third week of life and many more, perhaps a majority, by the beginning or middle of the second month. It is difficult to justify this practice on purely nutritional grounds. The infant's first nutritional need beyond that supplied by an adequate formula and vitamins is for iron and perhaps for some of the vitamins of the B complex, especially thiamine. Iron stores are sufficient for the first three or four months of life. Even these nutritional requirements could be met by medicinal administration.

"Some maintain that the early feeding of solid food is of psychologic advantage in that the baby can be accustomed to a wide variety of foods at an early age. It seems more likely that the practice is merely the result of competitive empiricism among both physicians and parents, a trend which began many years ago. It can be expected that the pendulum will shift back a little as more exact data are accumulated concerning the optimum time for introduction of solid food into the infant's diet. Considering the infant's nutritional requirements, three months of age would seem to be a reasonable time for routinely beginning solid foods. Exceptions can be made for rapidly growing, hungry infants who are not satisfied with full bottles of the milk feeding."

—LEE FORREST HILL, M. D.
in *Journal Lancet*, p. 74, Mar. 1956

CLEARANCE BY ALIMENTARY LUMEN CELLS

RICHARD P. SPENCER, *Lieutenant, MC, USNR*

METABOLITES and fluids ingested via the oral cavity are transported or "cleared" from the gastro-intestinal tract by cells lining its lumen. An evaluation of alimentary function is thus essentially a description of the workings of the luminal cells. Though we possess little information as to the composition of these units, we can infer much as to their activity and efficiency by studying the "clearance" of materials from the intestine to the body proper.

The term clearance (C) has been defined,[1] in renal physiology, by the equation

$$C = \frac{UV}{P} \qquad 1$$

where U and P are the urine and plasma concentrations of the compound, and V is the urine flow in ml/min. Thus, C is an expression of the number of milliliters of plasma that could be made free of the metabolite. Because there is no homogeneous fluid bathing each segment of the alimentary tract, this equation is not directly adaptable to an evaluation of gastro-intestinal function. Moreover, a single measurement cannot adequately represent transport from the alimentary lumen. However, even with present crude technics, several parameters can be evaluated in studying the complex phenomenon of gastro-intestinal clearance.

Static clearance (C_s) may be defined by the equation

$$C_s = \frac{Q_i - Q_e}{Q_i} \times 100 \qquad 2$$

where Q_i and Q_e are the quantities ingested and excreted. The clearance figure thus obtained neglects the time factor (any interval may be designated); but, more fundamental, it is "static," for it gives little insight into the dynamics of the luminal cells. Rather, it represents the percentage of the ingested load taken up.

In the daily average body water economy, upon drinking 2,500 ml, one fifth of this quantity may eventually be passed by way of

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the fecal stream, and C_s = 80 per cent. Hence, 80 per cent of the water was cleared by the gastro-intestinal tract (although because of turnover and endogenously produced water, none of the excreted molecules may be identical with the ingested ones). In diarrhea, if the same quantity of water were taken, and 1,500 ml passed, C_s would be 40 per cent.

Let comparative clearance (C_c) be determined by

$$C_c = \frac{C \text{ pathologic}}{C \text{ normal}} \times 100 \qquad 3$$

Comparing the diarrheal and normal states (40 per cent/80 per cent × 100), C_c is 50 per cent. Such a comparison would make it appear that in the diarrheal illness, luminal units were only 50 per cent as efficient as normal (possible mechanisms being rapid transit or cellular damage). However, even severe episodes of diarrhea do not reduce lumen cell function by as much as 50 per cent. This gross exaggeration tends to point out the difficulty encountered with a "static" figure, and brings us to a more dynamic evaluation of lining cell activity and efficiency.

Knowing the amount of product (P) brought to the alimentary lumen, and the material excreted (E) after a suitable time interval, the two terms are related by the expression

$$E = P + \Sigma \quad (p + s - a) \qquad 4$$

where the summation figure includes the amounts presented to (p), absorbed by (a), and secreted by (s), the individual segments. Knowing P and E, we can calculate the total function of all segments for any substance we choose, and under any indicated conditions (such as before and after the tract is cleansed of bacteria); or, by studying the product at different levels of the tract, we can evaluate the contributions of the segments above our reference point. Generally, the summation figure will carry a negative sign, as greater quantities of a metabolite are absorbed, or subtracted, from the tract than are eventually excreted by it.

The major difficulty with the static clearance figure (and unfortunately, this is all we have available at the present for most nutrients) now becomes apparent. C_s neglected the secretion of the luminal units in calculating the quantity of substance handled by the outermost portion of the lining cells. Equation 4 considered the function of the cell to be the uptake of materials appearing at the luminal surfaces, whether the compound came from the segments above *or from its own secretions.*

To illustrate: The 2,500 ml of water taken orally are but part of the fluid bathing the lining cells. For, in addition, the tract is also presented with an additional 8,200 ml by the secretory ac-

tivities of the salivary glands, hepatic system, stomach, pancreas, and intestine.[2] Active clearance (C_a), which represents the action of the luminal units on the total material presented to them, may be defined, during any specified time interval, by

$$C_a = \frac{(Q_i + Q_p) - Q_e}{(Q_i + Q_p)} \times 100 \qquad 5$$

where Q_i and Q_e are as before, and Q_p represents the total quantity of the compound added to the lumen by the secretions of the lumen cells or the appendages of the alimentary tract. This expression can be presented in the terms of equation *4* as

$$C_a = \Sigma \left(\frac{a}{p + s}\right) \times 100 \qquad 6$$

With a 2,500-ml water intake, an addition of 8,200 ml by the digestive organs, and a 500-ml fecal water loss, C_a would be 95 per cent. Hence, 95 per cent of the water is reabsorbed, an efficiency approaching that of the renal nephron. For the diarrheal illness previously discussed, C_a is 86 per cent. Comparatively, C_c is 90 per cent, not 50 per cent as determined by the static clearance concept of equation *2*, and we can infer that in terms of the total water presented to the lumen cells, some 90 per cent of normal is still absorbed in the pathologic state (assuming that the secretions of the appendages are unaffected by the diarrheal agent, or by substituting proper figures if they are altered). It will be observed that C_c is a comparison between a pathologic and a normal state, and can be determined for any clearance parameter defined.

Additional information can be obtained as to lumen cell clearance by considering the alimentary tract and its appendages as a single unit membrane with two functions: (1) filtration of plasma to form gastro-intestinal secretions; and (2) reabsorption of the filtrate plus ingested fluids and nutrilites. In such a system the clearance gradient can be defined as

$$C_g = \Sigma \left(\frac{a}{s}\right) \qquad 7$$

or

$$C_g = \frac{(Q_i + Q_p) - Q_e}{Q_p} \qquad 8$$

For the normal state discussed, C_g is 1.2 (that is, 1.2 times as much water is absorbed as is secreted), and in the diarrheal

episode, 1.1. Total clearance (C_t), or the total amount of product handled by the lumen cells in both directions, is simply

$$C_t = \Sigma (s + a) \qquad 9$$

$$C_t = (Q_i + 2Q_p) - Q_e \qquad 10$$

If cardiac output were 2,500 ml of plasma per minute, and this flow was distributed with 30 per cent to the alimentary tract and 20 per cent to the kidneys, over a 24-hour period typical values of the clearance gradient and total clearance would be as calculated in table 1. The percentage of flow filtered was determined from

$$\text{Percentage filtration} = \frac{\text{volume of secretion}}{\text{plasma flow}} \times 100 \qquad 11$$

TABLE 1. *Comparison of filtration and absorption factors in the alimentary tract and kidney*

Organ	Plasma flow ml/min	% flow filtered	C_g	C_t in ml per 24 hours
Alimentary tract	750	0.8	1.2	1.8×10^4
Kidney	500	20.0	0.99	27.3×10^4

When one considers that the renal apparatus is designed for "losing" fluid to the milieu exterieur, while the alimentary tract "gains," the clearance gradient values of 1.2 for the alimentary tract and 0.99 for the kidney, both so close to unity, loom all the more remarkable. They depict the narrow range within which homeostasis exists, and the function of the lumen cells in the maintenance of this equilibrium.

Such an analysis may be profitably employed in the study of disorders of alimentary tract transport (whether too great a clearance, as iron in hemochromatosis or copper in Wilson's disease, or too little clearance, as vitamin B_{12} in pernicious anemia). It also may have applications in the evaluation of materials which are recycled through the gastro-intestinal tract (as cholesterol and bile salts).

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THE FINAL COMMON PATHWAY IN ULCER GENESIS

JACQUES L. SHERMAN, Jr., *Major, MC, USA*

FROM 1942 THROUGH 1945 enough soldiers to have manned three combat divisions were separated from the Army of the United States because of gastric and duodenal ulcers.[1-3] The magnitude of the ulcer problem in the Armed Forces is further indicated by studies showing that the incidence of peptic ulcer in the Army is increasing.[4] It is said that from 5 to 10 per cent of the persons in most populations will develop an ulcer at some time in their lives.[5] In addition to a large morbidity, the death rate from gastric and duodenal ulcers is 5.6 per 100,000; more than 5 times that from poliomyelitis.[6]

This presents to medical scientists a challenge which does not appear to be capable of solution by better antacids or improved antispasmodic concoctions. The basis of current therapy still rests upon the empiric programs of the distant past: rest, "non-acrid diet," milk, and absorbent earths had all been prescribed in ulcer treatment by the seventh century A. D. Technics of administration of these agents have been modified, and by 1905 alkali powders and antispasmodic drugs were added. Despite these refinements the recurrence rate of peptic ulcer remains at least 50 per cent, and the prevalence of the disease continues to increase throughout the Western world.

The classical questions: "Why does not the normal stomach digest itself?" and "why is a single place in the stomach deeply affected when all the other parts of the organ are in a state of perfect integrity?" remain unanswered, despite tremendous efforts. However, too much attention has been directed to secretory functions, stress, the psyche, and pepsin, and too little to the gastroduodenal mucosa. As a result, little actual progress has been made since 1772 when John Hunter suggested that some "living principle" which depends upon circulation of blood prevents digestion of the stomach during life.[7] One need not be a heretic to wonder whether a fresh approach to the problem is in order or whether it is time for the "expurgation of the intellect," which Francis Bacon suggested, in order to get rid of fixed ideas which blind us.

From U. S. Army Hospital, Fort Belvoir, Va.

THE APPROACH

The concept of a final common pathway as a method of thinking about peptic ulcer may be helpful in applying some of the data already available and in clarifying some of the current concepts of ulcer genesis. Peptic ulcer is probably a local manifestation of a constitutional reaction; acid-pepsin is capable of digestion and excavation of injured gastro-intestinal mucosa. Nonetheless, the actual process of ulceration must be initiated by focal loss of viability of groups of cells within the intestinal wall. This concept is basic and one must accept it in order to understand such phenomena as the localization of duodenal and gastric ulcers to small regions of these organs and the tendency of ulcers to remain of fixed size. The thesis has been expressed in statements such as these:

"The stomach does not digest itself when its circulation is adequate, when the tolerance of the cells to acid-pepsin is not exceeded, and when the nutritional condition is adequate for the regeneration and proliferation of the cells of the gastric mucosa and for mucous secretion.

"The intestine does not digest itself when its circulation is adequate, or when the diluting, neutralizing, and buffering reactions between the gastric juice, Brunner's secretion, intestinal juice, bile, and pancreatic juice are normally maintained within the limits of tolerance of the mucosa to acid-pepsin and active pancreatic juice."[5]

". . . it is evident that the etiology of peptic ulcer disease in man should be sought elsewhere than in hyperactivity of the aggressive factor—rather in changes occurring in discrete areas of the gastric or duodenal wall . . . .

". . . it is conceivable that a deficiency may occur in the mucus-secreting activity of the columnar cells for metabolic or other reasons, e. g. an inadequate supply of one of the chemicals necessary for mucin synthesis or a diminution in the ability of the cells to effect this synthesis from the materials which are supplied them in the local circulation . . . .

"On the other hand, the balance between aggression and defense may be upset by discrete changes in the second line of defense. Such an upset would clearly result from any local physiological change which reduces the rate of reconstitution of the mucous epithelial layer after its impairment . . . . Reasoning back a step further, we ask what the factors may be which influence this rate of mucosal repair . . . . Among these are local ischemia of emotional origin, with its corollaries of localized anoxia, inadequate supply of nutritive elements, and the local accumulation of waste products of metabolism . . . ."[8]

"The keystone for a proper theory of ulcer genesis will be found in identification of the mechanism responsible for local depression of mucosal resistance. When there is spontaneous loss of substance from any

surface of the body, to form an ulceration, the basic cause of tissue dissolution is ischemia, whether the initiating process be infection, trauma, thermal influence or neoplastic change. Both clinical observation and experimental study have rendered it inconceivable that native gastric acid, however concentrated, could produce an ulcer under conditions of normal mucosal health."[2]

These quotations were selected because the authors are well-known proponents of the "acid-pepsin" theory, the "mucus barrier" theory, and the "ischemia" theory of ulcer genesis, respectively. All make reference to the importance of mucosal resistance, and it is the purpose of the concept of a final common pathway to focus attention on factors which lead to local depression of mucosal vitality.

THE FINAL COMMON PATHWAY

The characteristics of a final common pathway may be illustrated by the phenomenon of the contraction of fibers of skeletal muscle. When the complex protein actomyosin is exposed to adenosine triphosphate (ATP), shortening of the actomyosin fibrils occurs and skeletal muscle fibers contract. The final common path here is:

Actomyosin + ATP

This action is final since no other known event occurs before contraction; it is common because this path is used whether the initial stimulus arises in Betz cells, pain receptors, basal ganglia, or with the dropping of ATP on fibrils of actomyosin in vitro. A corollary of this concept is the inevitability of the event once the final path is activated.

This example illustrates the necessity for a careful search for a final common path (FCP), because the motor neuron is usually considered the "final common path" in muscle contraction. It is evident that this is far from being accurate, for there are several steps yet to be taken after stimulation of a motor neuron and before contraction. Also, a muscle fiber can be made to contract by chemical, thermal, or electrical stimulation independent of the motor neuron. Thus, the motor neuron is neither a final nor a common path. In addition, the stimulation of a given motor neuron need not cause contraction if transmission to the muscle is blocked, as with curare, for example. Thus its stimulation does not inevitably lead to muscle contraction.

The application of such a concept to the problem of the cause of peptic ulcer need not be difficult, although the actual elucidation of the pathway itself may be laborious. Starting with the premise that ulceration is initiated by focal devitalization of cells within gastric or duodenal mucosa, the search for an FCP begins

with a consideration of cellular damage. In general, important physicochemical effects on cells consist of: (1) solution or modification of the cell membrane; (2) change in osmotic pressure relationships; (3) alteration of electrical potential; and (4) alteration of the colloidal state of cell contents.

Obviously, agents or conditions which could produce any or all of these effects are numerous. Since the great majority of stomach and duodenal ulcers form in such restricted areas of these organs, we must seek a mechanism which can produce tissue damage and which can do so both locally and discretely.

THE MECHANISM

The three great physiologic systems which may be capable of this double action are the endocrine complex, the nervous system, and the circulatory system. The endocrine system is involved in the "stress" response and in the reactions of the gastro-intestinal tract seen in peptic ulceration. Gray et al.[10] have outlined one pathway by which these influences are mediated. But the endocrine disturbance, whatever its nature, is not capable of the fine, localized action necessary to explain focal devitalization of cells in specific areas of the stomach and duodenum. There are no known or suggested hormonal actions which could be an FCP leading to specific cellular damage; hormones are basically biologic catalysts and appear to be trophic only to endocrine tissues.

The nervous system, particularly the autonomic division, could provide either an FCP or a direct influence on one, for it is capable of response to generalized and varied stimuli, and it can direct actions to local areas. However, like the action of hormones, the effects of nerve impulses of the autonomic system are on end organs such as blood vessels, muscle fibers, and secretory glands and have no primary relationship to the viability of tissue.

The vascular system, like the nervous system, responds to general stimuli and can act locally. More important are the facts that its function is concerned specifically with cellular integrity and that its dysfunction may lead to rapid tissue death or loss of viability. The vascular system meets all of the requirements of a primary ulcerogenic pathway: it participates in generalized body responses; it is readily influenced by "stress" and by psychic phenomena; it has a degree of independence from other gastrointestinal functions; it is capable of local control and action; and it is specifically concerned with vitality of cells in all parts of the body.

In their review of the current status of a vascular basis for the ulcer process, Palmer and Buchanan[9] pointed out that it is just 100 years since Virchow and Rokitansky first incriminated the vascular system as the initiating mechanism of ulcer. During this time

many vascular theories have been presented but have not won acceptance because of their failure to demonstrate any occlusive mechanism or arterial disease which could explain focal devitalization. Recent anatomic and physiologic studies on the circulation have expanded the knowledge of the location, extent, and control of the vast system of arteriovenous anastomoses which exist throughout the body. This system is of such proportion that many of our concepts of vascular physiology and general pathophysiology must be revised. The presence of functioning arteriovenous shunts in the gastric and duodenal wall[11] has led to a new approach to the problem of the specific localization of ulcers, and we are at present carrying out investigations along these lines.

The final common pathway in ulcer genesis lies distal to the vascular system and may be conceived of as being a distortion of one of the normal functions of the circulation. Although it is tempting to assume that hypoxia* is the condition produced by circulatory disturbance leading directly to focal tissue devitalization, actual identification of the FCP still remains to be accomplished. Important functions, such as the provision of nutrients for function and repair, the removal of CO_2 and other wastes, the provision of substrate for enzymatic actions, the regulation of osmolarity, and others, need to be studied as possible components of a final path.

However, a satisfactory working hypothesis for the present is that hypoxia does represent the aberration of normal function which is an FCP in the genesis of ulcer. This hypothesis does not discourage investigation even more distally in the search for actions which are more closely related to the onset of tissue destruction. It is known, for example, that hypoxia causes the activation of native proteolytic enzymes in the cell.[12] This or other effects of hypoxia may be eventually identified as the actual FCP, and the hypothesis will have to be modified accordingly.

Supposing hypoxia to be the FCP, one may work centrally to establish the influences on the FCP. Disturbances of circulation, for instance, may lead to hypoxia by producing local ischemia or local stasis. Both mechanisms lead to hypoxia—the first to deprivation hypoxia, the second to "stagnant anoxia." In this way, it may be possible to understand some of the apparently conflicting observations on the presence of blanching or reddening of the mucosa in states associated with ulcer development. Thus, one may look for specific influences which can cause the production of local ischemia or of local stasis, such as nervous or neuro-

*The term "anoxia" actually means without oxygen, although common usage has given it the meaning of "oxygen want." "Hypoxia" designates a subnormal supply of oxygen and is used here in this general sense.

humoral effects on arterioles, arteriovenous anastomoses, or venules. In this way the concept directs a logical investigation of mechanisms of transmission of ulcerogenic influences and discourages simple classification of these influences as an approach to the "cause" of ulcer.

DISCUSSION

Recently a plea was made for "reason and imagination"[13] in the understanding of chronic peptic diseases. It is my belief that, unless reason is guided by the rules of logic, and unless imagination is confined by the requirements of the scientific method, we will not readily approach a better understanding of peptic ulcer. No explanation of ulcer genesis can be considered complete unless it can show how the responsible factors lead to localized cellular destruction in the gastric or duodenal wall, allowing acid-pepsin to digest the previously resistant tissues. The concept of a final common pathway immediately preceding this focal devitalization can help to clarify thinking in evolving any general theory of ulcer formation. Without doubt, psychic disturbances are important in the genesis of peptic ulcer, but it will serve little purpose to catalog various types of psychic dysfunctions without also attempting to trace these influences from the psyche to the FCP. This same requirement should apply to all theories of the causes of ulcer and the touchstone for any theory must be its ability to explain activation of an FCP. Such a requirement will direct attention away from the ulcerogenic influences themselves and will emphasize the mediation of these influences, an approach infrequently used in recent research.

The importance of the circulation in the various pathways leading to the FCP needs emphasis. In the foregoing quotations on causes of ulcer, each author indicated the opinion that, basically, the circulatory system could be responsible for the loss of defensive properties of the gastroduodenal mucosa. There are others with similar opinions who might have been quoted—authors who believe that the neurogenic influences are the most important, some who feel that allergic factors play an important part in ulcer genesis, and others who are impressed with the importance of constitutional factors. Much remains undone in a study of arteriovenous anastomoses in the gastro-intestinal tract, but enough information is available to indicate the fact that within this extensive system, which is under both neural and humoral control, lies the capability of great modification of mucosal blood flow. The flow of capillary blood seems to be under the control of these arteriovenous shunts; their size, number, and location allow them to act generally or locally and to regulate the rapid transfer of large amounts of blood within a motile organ system such as the gastro-intestinal tract. These characteristics fit so well the re-

quirements of a pathway which could activate an FCP that there has been a re-awakening of interest in the concept of a vascular basis for peptic ulcer.

SUMMARY

Peptic ulceration depends upon acid-pepsin digestion of a mucous membrane normally resistant to digestion. The major effort in investigation of the genesis of ulceration must be upon the factors that render this mucous membrane susceptible to such digestion. Since, generally, this digestion takes place in limited areas of the stomach and duodenum, these causative factors must be capable of exerting their effects locally as well as being able to produce tissue devitalization.

The concept of a final common pathway (FCP) has been outlined, proposing that some basic pathologic mechanism immediately precedes focal tissue devitalization, allowing acid-pepsin digestion to produce an ulcer in the region of the devitalization. A working hypothesis—that hypoxia is the FCP—is presented, and reasons are given for believing that circulatory disturbances are responsible for the localization and the hypoxia.

It is suggested that all theories of the causes of peptic ulcer be required to include possible mechanisms by which ulcerogenic influences are transmitted to an FCP. This may tend to guide and restrict thinking about causative factors in peptic ulceration and may help to clarify the reasons for phenomena that have been observed during this past century by clinician, research worker, physiologist, and pathologist.

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"The existing evidence indicates that the prophylactic use of antibacterial agents is of value under the following conditions:

1. In contaminated wounds of violence, including burns.

2. In elective surgical procedures performed through, or in, contaminated areas such as the gastrointestinal, respiratory, or genitourinary tracts.

3. In patients with associated derangements of the urinary tract who undergo surgical operations.

4. In patients with indwelling catheters.

5. In persons with pre-existing valvular heart disease who sustain injuries or undergo operations of the oral or pharyngeal cavities.

6. In patients undergoing emergency surgery in the presence of associated but unrelated infections such as acute tonsillitis.

7. For preparation of the gastrointestinal tract.

8. For elderly people with chronic pulmonary disease who undergo surgical treatment."

—WILLIAM A. ALTEMEIER, M. D.,
WILLIAM R. CULBERTSON, M. D.,
and MARK VETTO, M. D.
in *A. M. A. Archives of Surgery*
p. 5, July 1955

RECENT ADVANCES IN SUPPOSITORY MEDICATION

ANTHONY F. CACCHILLO, *Private First Class, USA*

A DOSAGE form that boasts a history as far back as the time of Hippocrates, and to which reference was made in Papyrus Ebers, is the suppository. From earliest times man has used this practical and convenient route of drug administration, with varying degrees of success. In the chemotherapeutic era of today, important opportunities remain for suppository therapy using new drugs. This method is particularly useful when other routes are unsuccessful, lacking, or impractical.

Advantageous localized treatment of the rectum, urethra, and vagina is accomplished when the drug is not absorbed by the mucous membranes. Emollient preparations indicated following surgical intervention of the rectum, in pruritus ani, vaginitis, and hemorrhoids provide satisfactory relief in this dosage form.

When systemic action is desired, those patients in whom oral administration produces nausea and vomiting and wherein intravenous therapy presents a problem are ideal subjects for routine suppository medication. This method of obtaining satisfactory blood levels is particularly well-suited and beneficial in: (1) Infants and children to introduce salicylates and other medicaments of therapeutic value. (2) The class of persons unable to swallow capsules or tablets, or in comatose condition and unable to receive conventional treatment. (3) The administration of drugs that are inactivated by digestive fluids of the stomach or intestine. (4) Those cases in which the drugs interfere with digestive functions.[1]

Information in the literature relative to satisfactory suppository bases, new formulas, and uniform dosage schedules is scanty and incomplete, and this valuable method of drug administration has not received its due attention and recognition. Only recently have great strides been made in the development of the suppository as a unique and thoroughly practical method of administering drugs.

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Blume and Nohara[2] reported that the absorption of sodium salicylate in rabbits is greater from the rectum than from oral ingestion. In another study[3] six drugs were tested for absorption in 63 normal persons. Five of these drugs, *viz.*, sodium salicylate, chloral hydrate, methylene blue, atropine sulfate, and morphine sulfate were absorbed in therapeutic quantities more quickly by rectum than when given orally. Other investigations[4, 5] revealed that higher concentrations of salicylate were obtained in the blood of human subjects by rectal than by oral administration.

A new type suppository base formulated by using varieties of Carbowax (brand of certain polyethylene glycols) has recently been cast into the medical-pharmaceutical spotlight. The characteristics of these compounds, of the general formula $HOCH_2(CH_2OCH_2)_x CH_2OH$, are such that undesirable properties of other type bases are excluded. They are unctuous, waxlike solids that dissolve in water, forming transparent solutions. They also are soluble in many organic solvents, do not hydrolyze or deteriorate on storage, and will not support mold growth.[6] Unlike cocoa butter, the polyethylene glycols require no refrigeration, and products of great pharmaceutical elegance which are esthetically acceptable to the patient can be produced by their use.

After having studied suppository bases and absorption via rectal administration,[1] I am confident that the polyethylene glycol type base will reach new heights of usefulness in medicine. To determine relative degrees of absorption, 11* male subjects on one day, for three successive weeks, were administered 0.64 gram of acetylsalicylic acid in suppository form in bases of cocoa butter, glycerinated gelatin, and polyethylene glycol (table 1). During the fourth week, two (0.32 gram) aspirin tablets were given orally. Plasma levels of salicylate were determined two hours after administration. Absorption from polyethylene glycol suppositories was found to satisfactorily approach that from orally administered tablets.

The purpose of this article is to bring to the attention of Medical Corps personnel recent great advances in suppository medication, and to invite a re-evaluation of its usefulness. It is hoped that presentation of the following formulas and compounding instructions will stimulate increased manufacturing by pharmacists and prescribing by physicians, with benefit to members of the armed services requiring medical treatment.

FORMULATION

These formulas are based on a standard suppository weight of 4.0 grams. The weight of the suppositories produced by the

*Results on two of these subjects were discarded because they could not retain the suppositories made with glycerinated gelatin.

TABLE 1. *Comparison of absorption by oral and rectal routes, using various suppository bases*

Age	Weight	Blood level mg per 100 ml			
		Polyethylene glycol base	Cocoa butter base	Glycerinated gelatin base	Oral tablets
44	128	1.0	2.6	1.7	4.6
21	130	2.8	2.6	2.2	3.0
22	140	3.6	3.65	1.0	4.8
29	143	4.4	2.8	1.9	3.0
22	148	3.4	2.5	3.0	3.4
40	152	3.65	0.65	1.6	3.2
30	154	2.8	1.2	2.6	2.6
20	180	3.8	1.90	1.6	3.8
21	190	4.0	2.8	1.2	3.2
Average blood level mg per 100 ml		3.27	2.30	1.86	3.51
Percentage of absorption*		93.1	65.5	52.9	100

*Compared to theoretical 100 per cent absorption by the oral route.

particular mold to be employed should therefore be determined, using a blank made of polyethylene glycols 6000, 1540, and 400 in 4-3-3 ratio. The amount of medicament in each formula can then be adjusted accordingly.

Acetylsalicylic acid suppositories

To make 75 suppositories; 0.65 gram (10 grains) *Grams*

Acetylsalicylic acid	48.7
Polyethylene glycol 6000	101
Polyethylene glycol 1540	75
Polyethylene glycol 400	75

Melt polyethylene glycols on water bath, being careful not to overheat. Add acetylsalicylic acid and stir until in a solution; pour into mold. Do not refrigerate.

Chloral hydrate suppositories

To make 75 suppositories; 0.33 gram (5 grains)

Chloral hydrate	24.4 grams
Polyethylene glycol 6000	110 grams
Polyethylene glycol 1540	83 grams
Polyethylene glycol 400	83 grams
Green color	2 drops

Melt polyethylene glycols on water bath, add chloral hydrate, and stir until in a solution. Add green color and pour. Do not refrigerate.

Aminophylline suppositories

To make 75 suppositories; 0.49 gram (7½ grains)

	Grams
Aminophylline	36.6
Polyethylene glycol 6000	105
Polyethylene glycol 1540	79
Polyethylene glycol 400	79

Make a paste of aminophylline powder with liquid polyethylene glycol 400. Melt remaining polyethylene glycols on water bath and add paste quickly with stirring. Do not overheat; pour. Do not refrigerate.

Pentobarbital suppositories

To make 36 suppositories; 0.13 gram (2 grains)

Pentobarbital sodium	4.68	grams
Polyethylene glycol 6000	55	grams
Polyethylene glycol 1540	42	grams
Polyethylene glycol 400	42	grams
Yellow color	2	drops

Mix pentobarbital sodium with polyethylene glycol 400 and stir until in solution; melt remaining polyethylene glycols, add, and pour into mold. Do not refrigerate.

SUMMARY

Recent developments in suppository bases, and the rationale behind increased usage of this route of administration are reported. Plasma level studies show that absorption of many drugs from polyethylene glycol suppositories compares favorably with absorption following oral administration. The advantages of this type base over other bases include: (1) greater degree of absorption, (2) nonrequirement for refrigeration, (3) complete stability, and (4) greater pharmaceutical elegance and patient acceptability.

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CHRONICITY IN "ACUTE" VASCULAR PURPURA

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ALTHOUGH vascular purpura (Schönlein-Henoch's Syndrome) is usually considered an acute condition, it has the potentiality for stubborn chronicity manifested by either a relapsing or an unrelentingly persistent pattern. It is our aim to present our experience with 6 patients during a two-year period in order to re-emphasize some of the chronic clinical aspects and to lend some support to the limited and judicious use of cortisone in the management of this disease.

CASE REPORTS

Case 1. This 35-year-old housewife had a 17-year history of multiple difficulties. In 1939, she developed episodes of diarrhea and sore throat accompanied by pain in the epigastrium at 2- to 3-week intervals. In 1941, she was diagnosed as having ileitis and had a laparotomy performed following which she was told that her right ovary had been resected for cysts. In 1944, some five years after the onset of her abdominal symptoms, she developed multiple joint pains with definite swelling and erythema of both knees. Epigastric pain usually accompanied the joint symptoms. During an episode requiring hospitalization in 1945, severe erythema nodosum appeared and was followed within one week by extensive erythema multiforme. Her temperature was 103°F. Initial skin involvement lasted five weeks and gradually subsided leaving "bluish spots" which persisted for several weeks. Late in 1945, for the first and only time, she developed typical purpura and petechiae in the popliteal fossae and about the elbows. There was elevation of temperature on this occasion. Then followed hospitalization in nine different hospitals over a period of almost two years.

Since 1947, she was treated on an outpatient status. She continued to have symptoms of joint pain, epigastric burning, bouts of diarrhea, and febrile attacks. She was given such diagnoses as rheumatic fever, rheumatoid arthritis, and myositis and also had two abdominal operations.

During these years her gastric analyses, gastro-intestinal roentgenographic series, barium enemas, cholecystograms, pyelograms, and electrocardiograms, roentgenograms of the peripheral joints, spine,

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and sacroiliac joints, urinalyses, blood cell counts, and numerous blood chemistries were, with the exception of a slight anemia which was reported on one occasion, within normal limits.

During the past two years, large raised erythematous plaques in the skin, preceded by a burning and stinging sensation of the involved areas, have accompanied her joint and bowel symptoms. A slowly mounting blood pressure, averaging 150/100 mm Hg, also had developed. The kidneys continued to remain free of involvement, as determined by studies of urinary sediment. A slight improvement in the joint and skin manifestations occurred during the summer months. However, she never was free of symptoms for any lengthy period.

Case 2. A 37-year-old housewife was first seen 24 months ago complaining of a purpuric rash on the lower extremities recurring over a period of 17 years. The onset occurred two weeks after an episode of antral sinusitis. The lesions began as pruritic wheals which then became hemorrhagic. They were always most marked about the lower legs, but occasionally extended upward as far as the waist. She noted painful swelling of the knees, elbows, and wrists with the more severe recurrences. She frequently had diffuse swelling of both legs. Throughout the course of her illness, she thought that exposure to cold aggravated her symptoms. In 1942, albuminuria and hematuria occurred for the first time, and has recurred from time to time since. At times she has had gross hematuria which she correlates with exposure to cold. In 1946, she was told at the Mayo Foundation that she had Schönlein's purpura. She slightly improved after 1952, which she attributed to the equable climate of Hawaii. However, she continues to have fresh purpuric manifestations from time to time, and to show evidence of renal involvement. She has been observed in many hospitals and clinics and has been told repeatedly that her platelet count was normal, and that she had a negative cold agglutination test. There was no other history of personal or family allergic manifestations. Syphilitic infection was denied.

Physical examination showed a healthy appearing woman. There were numerous large areas of brown pigmentation about the feet and ankles and extending halfway up the leg. There were several ecchymotic areas and slight edema about the ankles. The capillary fragility test on the arm was positive.

Bleeding time was 1 minute and 50 seconds; clotting time, 4 minutes and 50 seconds; platelet count, 165,000; hematocrit, 49 ml per 100 ml; white blood cell and differential counts, normal; and sedimentation rate, 2 mm per hour. The cardiolipin test was negative. Repeated urinalyses showed a specific gravity up to 1.030. Albuminuria was intermittently present up to 2 plus. At times the urine was grossly bloody and loaded with red blood cells, while at other times it showed no red blood cells on routine microscopic examination. There was no hemoglobinuria. Blood urea nitrogen and an intravenous pyelogram were

normal. A roentgenogram of the chest showed a partially calcified lesion, measuring 1 cm in diameter, in the left upper lobe of the lung.

Although a few scattered fresh purpuric spots were usually evident, the patient had a severe exacerbation of her purpura in April 1953 following a sunburn of the legs. This was accompanied by gross hematuria, 2 plus albuminuria, and a slight rise of her blood pressure to 130/100 mm Hg. In October 1953 she was treated with cortisone for three weeks with marked relief from the purpura and the swelling of the legs and ankles, but this treatment was not repeated because of concern about the nature of the lesion in the left lung. For the following seven months her symptoms were worse in association with her pregnancy as they had been with two previous pregnancies. Following pregnancy, she was admitted to the hospital for blood transfusion because of anemia. She was also started on a hypotensive agent for a slowly mounting blood pressure which had increased to about 150/100 mm Hg.

Case 3. A 21-year-old housewife was admitted to the hospital on 20 August 1953 with the complaint of generalized, nonitching urticarial lesions over the entire body for three weeks. Their intensity had been variable, but in general they were getting progressively worse. A sore throat had preceded the urticaria by about a week. This had been treated with penicillin; however the urticaria was already present when the drug was administered. The patient's past history suggested vasomotor rhinitis for about five years.

On physical examination the patient appeared healthy except for urticarial wheals over the entire body, but most marked on her lower extremities. Her temperature was 99.6°F. Blood cell count, platelet count, and bleeding and clotting times were all normal. The capillary fragility test was strongly positive. The sedimentation rate was 8 mm per hour.

Three days after admission, the skin lesions were definitely purpuric. The patient had not responded to antihistaminics and other measures and was therefore started on corticotropin (ACTH). The results were good, and within three days she had no new purpura except for one or two lesions on the legs. After a week of treatment she appeared well.

Two days after discharge from the hospital the patient had a recurrence of the urticaria associated with purpura—especially on the legs. She was treated with cortisone on an outpatient basis. The condition immediately responded, but relapsed when the cortisone was reduced to 50 mg per day. After about two months of receiving continuous outpatient care with antihistaminics and other medications, she was readmitted to the hospital. Her urticarial lesions were mostly on the shoulders and neck, but on this occasion was not purpuric. The lesions, while typically hivelike, did not itch. She was again treated

with cortisone and had only a partial remission but she had to leave the hospital because of transfer of her husband.

Case 4. A 47-year-old veteran was admitted to the hospital on 20 July 1953 with fever, nausea and vomiting, and severe abdominal pain radiating to the back. He had had innumerable similar attacks of a few hours to several days over a period of about 10 years. The location of the pain was variable, and at times was referred to his chest. The onset on several occasions was heralded by syncope lasting from a few minutes to a half hour. He had had hematemesis and melena associated with the pain on one occasion about six years previously.

Since 1938, he had been told that his blood pressure was high. He had mild wheezing, especially at night, for 10 years, interspersed with rare attacks of typical asthma. From 8 to 10 years he had recurrent mild aching and swelling of his joints, usually coinciding with the abdominal pain. From 5 to 6 years he had often noted slight pitting edema of the ankles in the evening. For 5 years he noted purpuric lesions. These would begin as tender, pruritic nodules which would develop into hemorrhagic areas several centimeters in diameter. Initially purple, they would gradually fade into yellow and then disappear. A small slough occasionally appeared in the center of some, leaving an ulcer which always healed promptly. He never had less than a half-dozen purpuric lesions at any time. He experienced episodes of giant urticaria, accompanied at least once with edema of the glottis, which responded to epinephrine while preparations were made for emergency tracheotomy. Most of the acute attacks were accompanied by fever, with temperatures of 102°-104°F, but he often had less severe fever and was usually able to work with a temperature as high as 101°F. He had been hospitalized many times, usually as an emergency, and had been extensively studied from the cardiovascular and gastrointestinal aspects. Diagnoses of peptic ulcer, gall bladder disease, psychogenic gastro-intestinal reaction, probable pancreatitis, angina pectoris, cardiac neurosis, and diverticulitis of the colon had been made on various occasions but none had been substantiated.

Physical examination showed a somewhat obese white man who appeared acutely ill. His temperature was 99.2°F; pulse, 100; and blood pressure, 190/110 mm Hg. His abdomen was diffusely tender with guarding and some rebound tenderness in the left lower quadrant. The retinas showed grade I hypertensive changes. There were occasional expiratory wheezes throughout both lungs and a 1 plus pitting edema of the ankles. In the skin of the trunk and legs, there were six to eight ecchymotic areas measuring from 3 to 6 cm in diameter and varying from purple to yellow in color. The capillary fragility test was positive.

The following studies were negative or within normal limits: roentgenogram of the chest, scout film of the abdomen, cholecystogram,

gastro-intestinal series, proctoscopy, repeated blood cell counts, platelet counts, bleeding, clotting, and prothrombin times, bone marrow smears, repeated urinalyses (maximum random specific gravity, 1.022), repeated blood cultures, L.E. cell preparations, urinary porphyrins, serum amylases, gastric analysis, cardiolipin, electrocardiograms, and a liver profile including a sulfobromophthalein sodium excretion test. Barium enema showed diverticulosis with no evidence of diverticulitis. Biopsy of a subcutaneous nodule and underlying muscle showed a lipoma and normal muscle.

During the first week in the hospital the patient remained acutely ill, with severe nausea and vomiting, temperature up to 102°F, and pain requiring opiates for partial relief. His blood pressure quickly stabilized in the vicinity of 140/95 mm Hg. Henoch's purpura was diagnosed at this point. He was treated with ACTH by intravenous drip which gave him complete relief of his symptoms within two days. His ankle edema disappeared and he lost six pounds in weight. Once treatment was started, no new purpura appeared, and the residuals of the old disappeared within 10 days. The patient stated that he felt better than he had for many years. One month later, after ACTH had been discontinued and while receiving 50 mg of cortisone daily, he had a recurrence of abdominal pain with severe diarrhea and passage of about a pint of red blood in his stools. The next day, giant urticaria appeared on his upper lip and around the eyes. All of these symptoms subsided within two hours after an additional dose of 100 mg of cortisone had been given.

After discharge from the hospital, his course continued much as it had previously. Attempts to manage his disease with prolonged cortisone therapy were hindered by a gradually increasing hypertension with blood pressures of about 200/130 mm Hg. He required a daily dose of from 125 to 200 mg of cortisone for effective control of his symptoms. It was therefore reserved for the more severe attacks, many of which were aborted by 100 to 150 mg taken on the patient's own initiative. He carried 250 mg with him at all times, which usually sufficed for one to two weeks' use. He continued to work effectively, often in the face of surprisingly severe symptoms, but still required hospitalization on two occasions during the past year, once with hematemesis. Antihistaminic and xanthine drugs did not seem to help him. Sympathomimetic drugs, particularly epinephrine and ephedrine, have seemed to be beneficial, but their action was inconstant and questionable.

He was readmitted to the hospital in May 1955 with a left hemiplegia which had its onset during sleep a few hours previously. This had been preceded for two weeks by increasing purpura and abdominal pain. His blood pressure on admission was 180/130 mm Hg, with a prompt drop to 150/90 mm Hg on bed rest, reserpine, and cortisone. Recovery from paralysis was almost complete.

Case 5. A 34-year-old housewife was admitted to the hospital on 10 December 1953 complaining of abdominal pain and swelling of the face, abdomen, arms, and legs.

Her history went back to 1930 when she noted swelling of the lips and abdominal pain following a mastoidectomy. With her first menstrual period in 1932, she had acute laryngeal edema which subsided while tracheotomy was under consideration. In 1939 she developed acute abdominal pain which was thought to be appendicitis. At operation, about a liter of clear yellow fluid was found in the peritoneal cavity. The proximal three feet of the jejunum were thickened, edematous, and had a reddened and slightly granular serosa. The appendix was removed prophylactically and she made an uneventful recovery.

In 1946, she began to have recurrent episodes of giant urticaria lasting from a few hours to several days. They tended to appear with her menstrual periods or with emotional tension. All parts of the body were subject to involvement, particularly the legs. She also sustained several attacks of acute laryngeal edema severe enough to require hospitalization. Also since 1946, she has had repeated attacks of severe, colicky, abdominal pain and swelling, occasionally accompanied by nausea and vomiting or diarrhea. There has been no bleeding except secondary to hemorrhoids.

In June 1947, the patient underwent emergency laparotomy in a civilian hospital for obstruction of the small bowel. The ileum was found obstructed and adherent to a granulomatous mass at the upper end of the old laparotomy scar. The mass was 3 cm in diameter and 1 cm thick, and showed "caseation" in the center. The peritoneal cavity contained a large amount of straw-colored fluid. The jejunum and proximal ileum were edematous and exhibited numerous 1-mm, white plaques on the serosa. Pathologic examination of the excised portion of the scar showed "nonspecific granulomatous tissue." A guinea pig inoculated with the ascitic fluid was negative for tuberculosis.

In December 1948, following an attack of severe abdominal pain, distention, and vomiting, an exploratory operation was performed for probable bowel obstruction. The peritoneal cavity again contained a moderate amount of straw-colored fluid. There was extensive edema of the jejunum, but no other abnormal findings were reported except for a very few fine adhesions. She made an uneventful recovery.

She was hospitalized at least a dozen times from 1947 through 1949 because of abdominal pain or laryngeal edema. In 1947 and 1951, during two pregnancies which were carried to term successfully, she had no manifestations of giant urticaria or pain. In 1952, a bilateral oophorectomy was performed, but the expectation that it might alleviate her symptoms was not realized.

For six months before her latest hospital admission, she had been followed as an outpatient in this hospital. She had continued to have bouts of angioneurotic edema or abdominal pain approximately every

two weeks, but the two symptoms were rarely present simultaneously. As previously, she has been treated with epinephrine in oil, antihistaminics, mercurial diuretics, intravenous calcium gluconate, and testosterone with some apparent benefit at times. A superimposed hyperventilation syndrome further confused the clinical picture.

Physical examination at the time of her latest hospital admission showed a well-nourished white woman who appeared only mildly acutely ill. There was marked localized edema of the face, abdomen, arms, and legs, and mild, generalized abdominal tenderness. A faint systolic murmur was heard best in the third and fourth interspaces just to the left of the sternum.

The following studies were within normal limits: roentgenogram of the chest, cardiolipin, urinalysis, stool smears and cultures, cholecystogram, gastro-intestinal series, barium enema, intravenous pyelogram, and fasting blood sugar. Her white blood cell count was 11,600 per μl with 82 per cent polymorphonucleocytes. Her hemoglobin was 12.1 g per 100 ml, and the sedimentation rate, 13 mm per hour. Her symptoms were not relieved by epinephrine, but were in three days by ACTH given by intravenous drip. She was discharged four days later.

During the next three months the patient was given cortisone orally in doses of from 25 to 100 mg per day. She continued to have edema off and on, but seemed to have obtained some relief from the abdominal pain on this program. She was readmitted at the end of this period for the purpose of discontinuing cortisone because of the impending transfer of her husband. On this occasion, she had edema of the left side of her face, left forearm, and both legs. She complained also of pain in the left ear, which the otologist attributed to vasomotor rhinitis and eustachian salpingitis. ACTH was given for 3 days after the cortisone was discontinued. There was no exacerbation of her symptoms in the process. She was given cortisone to carry with her for emergency use in the event of another attack of acute laryngeal edema, which had previously responded poorly to epinephrine, but dramatically to cortisone.

Case 6. A 29-year-old physician with a history of asthma and hay fever in childhood was admitted to this hospital on 28 January 1954. He was well until 3 weeks before admission when he had a mild upper respiratory infection for which he was not given antibiotics. One week later he first noted petechiae on his ankles associated with burning epigastric pain unrelated to meals. Two days later his knees, ankles, elbows, and left metacarpophalangeal joints became red, painful, stiff, and swollen. Six days before admission an effusion of the left knee was tapped. A clear sterile fluid was obtained. Initially he was treated for four days with 300 mg of cortisone orally with excellent symptomatic improvement. However, when the medication was stopped, all of his symptoms recurred and, in addition, the calf of his left leg became swollen and tender.

Physical examination showed an alert patient who was not acutely ill. His temperature was normal. There were numerous petechiae about both ankles as well as several larger ecchymotic areas. The ankles, knees, and left elbow were swollen, tender, and had limited motion. The white blood cell count was 16,400 per μl with a normal differential count. The sedimentation rate was 28 mm per hour. Electrocardiograms on two occasions were normal. The kidneys seemed normal.

Cortisone was reinstituted at 300 mg per day with a gradual reduction to 100 mg per day for two and one-half weeks, followed by ACTH for one week. The patient had lower abdominal cramping pain and diarrhea for the last three days of his reduced cortisone treatment. Two days after the ACTH was discontinued, he had a mild flare-up of joint pain which lasted only a few days. After three weeks of treatment because his symptoms and findings subsided and he appeared well he was discharged from the hospital. A month later, he had a more severe recurrence of joint pain and petechiae which necessitated re-treatment with cortisone for about one month without loss of time from duty. He remained well for the past year.

CLINICAL ASPECTS

As in the acute, self-limiting form, the chronic clinical manifestations are diversified not only in different patients, but at different times in the same patient. An awareness of these pleomorphic qualities makes the disease more common than is generally appreciated. Our two-year experience reported here does not differ from an occurrence rate standpoint with previous experience. Osler[1-3] originally, and later Pratt,[4] emphasized and completely documented the chronic potential and the extremes in clinical variations in this disease.

Skin and Mucous Membranes. Of the systems involved, the skin perhaps offers the widest spectrum of variability of lesions. Urticarial wheals with or without itching may occur unaccompanied by or interspersed with other lesions. The itching, if present, is usually slight and sometimes described as a burning or stinging sensation just prior to the actual appearance of the wheal. Purpura may vary from scant and discrete embolic-type lesions level with the surrounding skin to relatively massive exudativum multiforme involvement with central areas of purpura and necrosis and a tendency to coalesce. Localized areas of angioneurotic edema may also appear initially or at any time during the course of the disease.[1-4] This potentiality was illustrated by cases 4 and 5. From the standpoint of duration, lesions may be minimal and transient throughout the entire course, or may come in successive crops if the disease waxes and wanes in a chronic relapsing form. In some instances it is possible to note areas of brownish pigment, sometimes even large coalescing areas (case 2) when the skin has been significantly involved

but not recently active. In any patient with pigmentation of this type not readily explainable by the more common causes, the possibility of vascular purpura should be considered.

Capillary fragility tests are unpredictable and apparently not always related to the severity of the purpura. The test may be positive in patients with mild skin involvement and negative in those with extensive purpuric lesions. There does seem to be a tendency, however, for the skin manifestations to be greatest in the dependent portions or over the pressure areas of the back. Ackroyd[8] mentioned that bright red lesions in some instances appeared 12 to 24 hours after slight local trauma such as stroking with a blunt rod or intradermal injection between attacks.

Gastro-intestinal. The gastro-intestinal component is generally manifested by abdominal pain of a colicky, sometimes burning type varying in severity, duration, and location. There usually is vomiting and occasionally hematemesis with appreciable blood loss. Bouts of diarrhea sometimes constitute a prominent complaint, and bloody stools and melena can be exsanguinating. The severity of the gastro-intestinal manifestations may suggest intestinal obstruction, perforation of a viscus, intussusception, pancreatitis, appendicitis, peritonitis, and other acute intra-abdominal situations, resulting in unnecessary surgical exploration. The lesion most commonly found at operation was an extravasation of blood or serosanguineous fluid into the wall of the small intestine.[5] In one of our patients (case 5), the surgeon stated that he found a "long section of edema of the bowel."

In the chronic form, the gastro-intestinal pattern is one of recurrent acute episodes often synchronized with joint and/or skin manifestations. The attacks are usually brief and self-limited but may also occur in rapid succession and of sufficient duration to cause total incapacity. The concern of the patient over repeated undiagnosed abdominal crises, and the subjection of these patients to unnecessary surgical exploration can be minimized only by developing an awareness of this disease in both its acute and chronic aspects. Reportedly an occasional patient required surgical intervention because of hemorrhagic ulcers with perforation or intussusception with obstruction.[5] However, Pratt, in his extensive review, cited the occurrence of only one case of intussusception. The gastro-intestinal component may occur with, recur with, or even antedate the onset of all other manifestations. It may occur simultaneously with combinations of joint, skin, renal, or other less frequently involved organ or system. A careful history will help more than any other single factor when attempting to fit together the manifestations in joint, skin, and gastro-intestinal tract that occur as apparently unrelated single episodes, or when the patient stresses only the most troublesome involvement to the exclusion of other features.

Joints. Joint manifestations while usually present may be minimal or absent. Some patients complain more bitterly of pain in the muscles rather than joints, particularly in the lower leg. The clinical spectrum of joint involvement is wide indeed. Only a single joint may be involved or there may be a migratory, extensive, polyarticular involvement. The intensity varies from slight arthralgia to severe, totally incapacitating pain with hot, swollen periarticular tissues and reddened skin. The pain may last over a prolonged period with some waning of intensity at intervals, but with discomfort more or less constantly present. However, a relapsing course of variable severity for each attack constituted the more common pattern of the chronic type of this disease. For the most part, the joint manifestations accompany other features of the syndrome. However, joint symptoms may antedate the other manifestations or occur as an isolated feature during any single attack. Once again, careful history will assist in uncovering a fleeting erythema or purpura, an attack of nonitching urticaria-like lesions, or repeated episodes of abdominal pain or burning. Suspicion aroused sufficiently by an unexplained arthritis may lead to recognition of the correct diagnostic pattern, particularly in the more chronic cases which have a longer time in which to develop all the features of the syndrome.

Renal. Renal manifestations or complications in the form of glomerulonephritis occur in a substantial number of patients, and Philpott and Briggs[6] reported as high as 47.5 per cent in children. There may be either gross or microscopic hematuria with casts and proteinuria.

The kidney may show involvement at any time during the evolution of the syndrome, most commonly appearing as acute nephritis followed by apparent complete recovery. However, serial urinalyses over a prolonged period may reveal cyclic recurrences of abnormalities on urinalyses suggesting a continuing or intermittent pathologic process in the kidney (case 2). The outlook for this latter group may be no better than for those who have chronic progressive nephritis.

Fever. Fever is usually present but may not be a prominent feature during a prolonged course or during any single acute exacerbation. Temperature elevation to 104°F (case 5) is common. Some patients may have classical involvement of the joints and skin with little or no temperature elevation. As a general rule, however, fever tends to accompany exacerbations in more severe chronic forms.

Other Organs Involved. Due to the angiitic nature of the other organs and systems may become involved. Lar[illegible]ge. occurred in two of our patients (cases 5 and 6) a[illegible] has reported by others.[1-4] Three of our patients dev

tension (cases 2, 4, and 6). In one of these (case 4), a recent cerebrovascular episode was immediately preceded by increasing purpura and bouts of abdominal pain.

TREATMENT

The treatment of this disease leaves much to be desired. Until the advent of the steroids, most therapeutic measures offered nothing superior to the passage of time except for an occasional patient in whom there was claimed to be an etiologic relationship to some specific allergenic agent. It is generally accepted that the steroids do have a favorable effect, at least on the immediate acute symptoms of the disease.[7-10] However, this method of management is not entirely satisfactory inasmuch as lack of benefit has been reported, particularly in regard to renal involvement.[6,11,12] Cortisone as utilized in our patients was not instituted for its permanent curative value, but employed as a temporary modifying agent similar to its use in bronchial asthma, collagenous disorders, and other disease states. It may add something of value to the management of the patient even if only to influence favorably the acute recurrent episode arising in the chronically afflicted person. The utilization of cortisone and ACTH in the care and management of these patients should be individualized, according to the degree of disability, intial response, and subsequent course. It is possible under favorable circumstances to convert an invalid to one who can be gainfully occupied (cases 1, 4, and 5), and even to save the patient's life during an acute crisis such as laryngeal edema (cases 4 and 5).

SUMMARY

Acute vascular purpura is not always an acute self-limited process. Chronic potentialities do exist and must receive adequate consideration in differential diagnosis. This is particularly true in those instances where the classical joint and skin manifestations are not striking in any single attack, and may be dissociated from gastro-intestinal complaints and from each other. It is only by careful consideration of all features presented by a series of separate attacks that the true diagnostic picture becomes evident. The apparently generally accepted concept that vascular purpura is an acute self-limited process should be abandoned inasmuch as a significant number of cases fall within a chronic disease pattern. This feature received adequate consideration in the original descriptions of the disease by Osler. However, in subsequent medical writings, while occasionally mentioned, it has received insufficient emphasis.

Once the diagnosis in this sometimes baffling chronic state is made, the physician is still confronted with the difficult problem of management. Other than steroid therapy, there remains little the way of effective symptomatic treatment. Even

the results with steroid therapy may not always be satisfactory or predictable. This is particularly true of the renal complications. Nevertheless, steroid therapy does alter the manifestations of the disease favorably to a limited extent and may permit one to treat the patient during the most troublesome phases, perhaps to an extent consistent with continued gainful employment. If the basic concept of a diffuse process incident to a small blood vessel angiitis on an immunovascular basis is accepted, then one is immediately aware of the multiple organ involvement.[13,14]

The generally favorable modifying effect of the steroids adds support to the hyperimmune concept of this vascular disorder. Lecutier[15] has likened vascular purpura to periarteritis nodosa. Dameshek[13] reviewed the evidence favoring this concept and drew the analogy between this disorder, thrombotic thrombocytopenic purpura, and disseminated lupus erythematosus.

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Clinicopathologic Conference

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HEADACHE, LOW BACK PAIN, AND DIPLOPIA

Summary of Clinical History. A 20-year-old white male airman entered the hospital for the first time on 23 September 1952, with the chief complaints of headache and decrease of vision for approximately two and one-half months. The patient was apparently perfectly well until about 4 July 1952, when he noted the insidious onset of headache, which started in the region of the orbits and very often involved the entire calvarium. These headaches came on at any time of the day and seemed to persist despite the usual analgesics. About a week later he noted also the insidious onset of low back pain with no definite radiation into the legs, which persisted until admission.

By about the end of July 1952, the patient started experiencing diplopia, worse on looking down and to the left. In early August 1952 he experienced several days of febrile illness vaguely described as "flu," associated with some general malaise, for which he was given streptomycin sulfate. This apparently cleared and he was able to return to duty. About three weeks prior to admission, he noted the beginning of progressive visual acuity difficulties such as blurring and indistinctness. At the time of admission, headaches were less severe and seemed to be associated with pain behind the eyes; he had a suboccipital soreness, but denied trauma, loss of consciousness, seizures, or sphincter

*At the time of this conference, Col. Robert S. Brua, USAF (MC), was Commanding Officer. From the Laboratory Service, Capt. Thomas J. Cooper, USAF (MC), Chief at that time.

control problems, and other disabilities such as ringing in the ears, difficulty in swallowing, or unsteadiness of gait. His memory had been unimpaired.

The patient had been in military service for 17 months, and had always been in this country. Prior to entry into the service, he had been a student. The remainder of his past history was noncontributory.

Physical Examination. Physical examination revealed a well-developed, well-nourished, mentally clear white man in no acute distress. Blood pressure was 130/75 mm Hg; pulse, 78 per minute; and temperature, 98.6°F.

Examination of the head revealed no skull deformity and no audible bruits. The neck was supple, with trachea in the midline, with no tracheal tug, and the thyroid was not palpable. Carotid pulses were strong and equal.

Examination of the eyes revealed the left palpebral fissure to be slightly larger than the right so that more of the sclera on the left was visible. Conjunctivae and sclerae were clear. The extra-ocular muscles showed no objective change; however, the patient claimed that there was diplopia on looking down, and especially to the left. Visual fields were grossly normal. The ears, nose, throat, and mouth were not remarkable.

The lungs were clear to percussion and auscultation. The heart was not enlarged and had a regular sinus rhythm. The aortic second sound was louder than the pulmonic second sound, and there seemed to be a split first sound at the aortic area; however, no murmurs or friction rubs were heard.

Examination of the abdomen revealed no tenderness or hernias, and the genitalia were normal. On rectal examination, sphincter tone was good, and no masses or blood were noted. The prostate was normal.

On examining the back, no limitations of motion were noted; however, there was a peculiar deformity in the upper lumbar or lower thoracic area, which seemed to be asymmetrical on flexion with a concavity appearing on the left of the spine and a convexity appearing on the right. No pain in the back resulted on straight leg raising. There was no cyanosis or edema of extremities. Both femoral and popliteal pulses were felt, but neither the dorsalis pedis nor posterior tibial pulses were felt.

Neurologic examination showed the patient to be right-handed. His gait was normal, but on testing muscle strength, there was weakness of the plantar flexors on the left with no evidence of rigidity, atrophy, or muscle tenderness. Muscle strength and volume tone were normal elsewhere. Co-ordination was good.

Position sense was normal. Stereognosis was normal. No abnormal associated movements were noted. Reflexes were normal, except that the abdominal reflexes were 2 plus in both quadrants on the right and only 1 plus in both quadrants on the left. In the examination of the cranial nerves the olfactory sense was not tested but was described by the patient as satisfactory. Visual acuity seemed to be depressed; there were 2 to 3 diopters of papilledema in both eyes but no evidence of hemorrhage. On examination of the extra-ocular muscles, the patient complained of diplopia, and when the object presented was in the lower field of vision the image seen from the right eye was a good deal lower than that seen from the left. Corneal reflexes were intact. There was no evidence of motor deficit in the mastication muscles. The remainder of the cranial nerves were normal. Sensation to pin prick was normal. The patient's speech and mentation seemed to be adequate.

Laboratory Studies. Hemoglobin was 13 g/100 ml; the white blood cell count was 10,850/μl with 70 per cent neutrophils, 4 per cent polymorphonuclear leukocytes, and 26 per cent lymphocytes. Coagulation time was 3 minutes and 40 seconds. Urinalysis revealed a specific gravity of 1.020 and was negative for albumin and sugar; there was 0 to 1 white blood cell per high power field.

On electroencephalography an excellent technical tracing revealed a well-developed alpha activity in all areas at a frequency of 10½ cycles per second and reached an amplitude of 100 microvolts in the occipital areas. The bipolar records, both anteroposterior and transverse, showed no abnormal wave forms or frequencies. The monopolar linkages likewise showed no abnormality on the regular run. However, after 1½ minutes of hyperventilation, a marked breakdown in the pattern sets, characterized by long bursts of delta potentials at 2½ cycles per second in all leads, occurred. These reached a very high amplitude, sometimes as much as 400 microvolts. In some runs the characteristic "spike and dome" complexes were noted. This breakdown in the normal pattern persisted for some 25 minutes after the cessation of the overbreathing.

Visual fields revealed a slight, generalized constriction of questionable significance.

Course in Hospital. A ventriculogram was done on 26 September under local anesthesia and 45 ml of air injected. Filling was good except that no filling of the fourth ventricle occurred. There was no evidence of shift, and the slight deviation appeared to be symmetrical. The postoperative condition of the patient was good. Attempts at filling the posterior fossa from below were unsuccessful. On 28 September, the ventricle was tapped on the left side and fluid was released under great pressure. The patient

complained of his eyesight becoming progressively worse, but his general condition remained good. On 29 September, a Torkildsen procedure was done. During the course of induction the left lateral ventricle was punctured because of the patient's rather shallow respirations. Neither lateral ventricle could be located with a ventricular needle, after a few minutes. A right occipital flap was turned down and the right occipital lobe was removed in order to see into the ventricle. The right lateral ventricle was entirely filled with clotted blood; when this was removed it could be seen that the third ventricle was also filled with a blood clot. No further procedure was carried out and the wound was closed.

After return to the ward, the patient suddenly had a high elevation of blood pressure (200/120 mm Hg) and pulse rate (240 per minute). A ventricular tap was done, revealing grossly bloody fluid under great pressure. Attempts at introduction of a polyethylene and a No. 10 French catheter into the left lateral ventricle were unsuccessful. An endotracheal tube was re-inserted and respirator applied. Blood pressure dropped to 90/65 mm Hg, and pulse remained high at 200 per minute, the patient still breathing spontaneously at 1 to 2 per minute. At 1930 hours that same day there was no further change in his condition except that blood pressure varied from 210/100 to 80/40 mm Hg. Neosynephrine (brand of phenylephrine hydrochloride) was given by continuous intravenous drip. The heart was not enlarged on physical examination and heart sounds were of good quality, but the rate was 140 per minute with regular rhythm. The patient was then prophylactically digitalized with digitoxin intravenously, because of a pulse rate varying between 120 and 240 per minute for the previous 5 to 6 hours. At 2115 hours, 4 ml of Cedilanid (brand of lanatoside C) intravenously and 50 mg of Neosynephrine by continuous drip were given.

By 2330 hours, the blood pressure had stabilized at 90/70 mm Hg, and the pulse dropped to 150 per minute. The patient, however, looked worse clinically; the pupils were widely dilated and did not react to light, the patient being in a deep coma. Heart sounds were not of good quality, but still no signs of cardiac failure had appeared. Four milliliters of Cedilanid were then administered. By 1700 hours, on 30 September, all reflexes, as well as neurologic function had disappeared. On 1 October, at 0600 hours, the patient was pronounced dead.

DISCUSSION

Doctor Baker:* The essential features of this 20-year-old airman's history were the insidious onset of orbital and generalized headache 12 weeks prior to hospitalization, with low back pain, diplopia, and

*Capt. Robert N. Baker, USAF (MC), Neurology Service.

a transient episode with "flulike" symptoms occurring during the first month of illness. Except for a few days he was well enough to continue his regular duty. Three weeks prior to hospitalization he noted progressive blurring and indistinctness of vision followed by diminution in severity of headache and associated with pain behind the eyes and suboccipital soreness. Physical examination was remarkable for its normalcy. A thoracolumbar scoliosis and absent distal lower extremity pulses were reported. Neurologic examination showed bilateral papilledema and diplopia most marked on looking down and to the left. An inequality of the palpebral fissures was also noted. The headache in this case was nonspecific in type. Its onset was insidious, periorbital, and often generalized. We do not have the details as to its intermittency or relationship to activity or time of day, but we are told that they were less severe by the time he was hospitalized. At that time they were associated with pain behind the eyes and suboccipital soreness.

The general features of this headache are those seen in many systemic illnesses as well as in many nonfocal disorders of the nervous system and therefore do not contribute much to the localization of an intracranial process in this patient. In any neurologic condition the initial location of head pain or headache is of interest to us because it may prove a factor in lateralization. This usually is the case only in the early stages before the headache becomes generalized, or before increased intracranial pressure itself obliterates the localizing features. In this case the periorbital pain is quite nonspecific. Not only can any intracranial process above the tentorium produce this type of pain, but so can increased intracranial pressure alone as well as entirely nonspecific toxic tension factors.

A week after the onset of this low grade headache, the patient experienced insidious onset of low back pain. We are not told whether any particular stress such as activity, coughing, or posture affected this pain. However, we learn later that there was a deformity in the thoracolumbar area, resulting in scoliosis on flexion of the spine. There were no local signs nor evidence of nerve root compression on examination. The bony deformity was most likely unrelated, but the backache could either be related in a nonspecific way or represent a mild meningitic reaction, possibly to blood, infection, or neoplasia.

The next feature to be considered is the early onset of diplopia. This began approximately two weeks after the onset of the headache and preceded by a week the onset of the febrile illness in early August. From the onset, it was a fairly specific type, that is, worse on looking down and to the left. This was the same picture that presented nearly two months later at the time of his admission. However, by the time of admission the patient had another complaint, that of progressive visual acuity difficulties, with blurring and indistinctness of vision. However, as it is reported in the clinical summary one has the impression that this is a separate phenomenon. We would like to know,

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however, whether the blurring occurred in both eyes, and if the patient thought it was a different affair from his diplopia. I mention this because in many cases a patient who states that he has blurred vision is actually experiencing this on the basis of extra-ocular muscle imbalance or paresis, and it is not due to interference with the visual pathways. In this instance, however, it seems safe to assume that this is a separate complaint related to the papilledema. The statement that the extra-ocular muscles showed no objective change probably indicates that there is no observable strabismus to simple direct inspection. With the distinct diplopia on looking down and to the left it seems likely that with the use of a light and colored lens, one could have objectively demonstrated this disorder. A detailed extra-ocular muscle analysis would have been very helpful.

It was also stated: "Examination of the eyes revealed the left palpebral fissure to be slightly larger than the right so that more of the sclera on the left was visible." This may mean that the abnormal fissure is on the right and that there is ptosis of the right lid due either to a third nerve, levator palpebrae weakness, or to interruption of sympathetic fibers on that side with loss of tone of orbicularis oculi muscles. Or it may mean that the abnormal fissure is on the left with widening consistent with a partial seventh nerve paralysis on the left. Since there was no description of difficulty in closing the eye or other left facial weakness, this latter possibility seems quite unlikely. We should also mention the possibility of mechanical distortion by something in the retro-orbital region pushing the globe forward. No description of exophthalmos is included, however. In any analysis of unequal palpebral fissures, one of the most important observations is the state of the pupils. If the right ptosis is associated with a dilated pupil the lesion would clearly be that of a third nerve parasympathetic paralysis. On the other hand, if the ptosis was associated with a small pupil one would think that a sympathetic palsy on the right would be more likely. Do we have a report on the pupils?

Doctor Strong:* The first time the pupils were mentioned was postoperatively.

Doctor Baker: That observation of course would be very valuable in our analysis, but we will have to do without it. Let us return to the diplopia. Whenever the patient claims there is diplopia only on looking down and especially on looking to one side, there is a good likelihood that the disturbance is in the fourth or trochlear nerve. In such lesions the diplopia is maximum when the involved eye is rotated medially. In this instance this would fit a superior oblique paralysis on the right side. The only alternative would be that of partial third nerve palsy or a combination of palsies. In most instances, however, third nerve lesions produce more complex extra-ocular imbalances. It would be quite rare to find one that simulated a trochlear lesion alone; on the other hand, the right-sided ptosis suggests a third nerve lesion

*Capt. Jack P. Strong, USAF (MC), Pathologist.

on the right also. I do not believe we can settle this matter finally. The presence of diplopia, however, does tell us that there is a lesion either involving the peripheral extra-ocular nerves or intrinsic to the midbrain itself. Occasionally something involving the muscles themselves may simulate a neural lesion. This is uncommon and does not fit here. The extra-ocular muscle findings do tell us that the disturbance is at the base of the brain, and very likely at the level of the midbrain or at the incisura tentorii. As noted before, the evidence suggests that the lesion is more likely to be on the right than on the left.

Turning to the impairment of visual acuity this would seem to fit quite well with the findings of papilledema. The slight constriction of the visual fields is also consistent with papilledema. The fact that papilledema seemed symmetrical in the two eyes does not exclude a lateralizing lesion but is consistent with intracranial pressure of any type, including an expanding lesion at the base or in the posterior fossa. The characteristics of the headache are consistent with those seen in increased intracranial pressure.

Most significant in this case is the absence of any other striking neurologic abnormalities. There is nothing in the examination to suggest a focal cerebral lesion. The statement that plantar flexion on the left was weak probably refers to a difference in the response of the great toe to plantar stimulation; that is, to a nascent Babinski sign on the left. The presence of a left Babinski would fit quite well with a right midbrain lesion. The only reflex abnormality was the abdominal reflexes which were diminished on the left. This also would go with the possible left Babinski and right extra-ocular muscle signs. The cardiac murmur and absent dorsalis pedis pulse do not seem related to his central nervous system disorder.

The laboratory data are not helpful. A spinal was not done, undoubtedly because of the evidence of increased intracranial pressure. In a problematic case such as this I would have done a spinal with removal of only a small amount of fluid. The electroencephalogram is of interest: During hyperventilation, symmetrical bursts of 2½ cycles per second waves reaching an amplitude of as much as 400 microvolts were seen. In some runs these appeared as "spike and dome" complexes. This is a significantly abnormal response and is suggestive of a deep lesion. But there is no electroencephalographic evidence of laterality. It is consistent with a disturbance at the base of the brain.

We have a gradually progressive central nervous system disorder with headache, extra-ocular muscle abnormalities, and papilledema. In addition there was a brief febrile episode and mild backache. Although an infectious disease cannot be entirely excluded in this case, it seems much less likely than an expanding intracranial lesion. Such an expanding lesion, either intra- or extra-medullary—in the region of the posterior third ventricle, around the tentorial incisura, or in the

posterior fossa with pressure in the region of the midbrain—seems most likely. Among the expanding lesions we must consider neoplasia, vascular anomalies, and foci of infection. This may be a rare glioma of the midbrain. It could be an ependymoma of the fourth ventricle with midbrain pressure and possibly seeding to the site of the spinal pain. An astrocytoma of the cerebellum may show itself only by pressure signs. In this case there could be early infiltration into the midbrain.

To mention the infectious possibilities: First would come a granulomatous meningitis with occlusion of the foramina of Luschka and consequent internal hydrocephalus. Occlusion of penetrating vessels at the base might account for focal midbrain signs, et cetera. Another rare possibility would be a focal granuloma or abscess acting as an expanding lesion. In this group a tuberculoma or a fungus granuloma should be mentioned. My first choice, however, will be a vascular anomaly such as an arteriovenous malformation at the level of the tentorial incisura. Such a lesion could act first as an expanding mass, secondly account for the headaches and backache, and possibly contribute toward the febrile episode. It would most readily account for the confusing sequence of events which followed the patient's hospitalization and led to his death. A vascular meningioma would fit the picture also. My diagnosis will be: first, vascular tumor, and second, possible midbrain or cerebellar glioma. Its location would be at the level of the midbrain or the incisura tentorii and most likely more on the right side.

Let us now turn to the course in the hospital. A ventriculogram was done on 26 September under local anesthesia and 45 ml of air injected, which is slightly more than average. What do you think, Doctor Mendelsohn?

Doctor Mendelsohn:* You don't want to know how much air was injected, you want to know how much fluid you got out. You have an open system and if you inject 100 ml with a capacity of only 20, it goes in one side and comes out the other. It would be much more helpful to know how much fluid was drained. Here it was probably the same. The normal volume of fluid in the ventricles is from 20 to 30 ml.

Doctor Baker: Filling was good except that no filling of the fourth ventricle occurred. In other words, they apparently filled the third ventricle adequately. Of course, it is very difficult sometimes to fill the fourth ventricle. It doesn't prove that the trouble was back in the fourth ventricle, although it is more likely since the ventricles were all right. There was slight dilation of the lateral ventricles which appears to be symmetrical. The postoperative condition of the patient was good. Attempts to fill the posterior fossa from below were unsuccessful and this is probably of significance, although sometimes it is hard to do. On 28 September, the ventricle was tapped on the left side and fluid

*Maj. Robert A. Mendelsohn, USAF (MC), Chief, Neurosurgery Service.

was "released under great pressure." I assume that this meant that the fluid appeared normal and not tinged with blood. Subsequently the patient complained of his eyesight becoming progressively worse, but his general condition remained good. On 29 September a Torkildsen procedure was done.

Doctor Strong, can you tell us whether or not there was a lapse of time between the Torkildsen procedure and the exploratory craniotomy?

Doctor Strong: Apparently the Torkildsen was accomplished, but subsequently the patient did not do well and he was again taken to surgery.

Doctor Baker: It seems, then, that the first ventricular puncture on the 29th was normal, but that during the second one they couldn't locate the ventricle. A right occipital flap was turned down and the right occipital lobe was removed in order to see into the ventricle. The right lateral ventricle was entirely filled with "clotted blood." When this was removed it could be seen that the third ventricle was also filled with a blood clot. No further procedure was carried out, and the wound was closed. In trying to decipher just what happened, I would say that something apparently happened between the successful ventricular tap and the time they opened the ventricle and found the clotted blood. In the light of these events I wonder if this was not a primary vascular lesion of some sort around the incisura tentorii, that the disturbance of intracranial dynamics at operation resulted in hemorrhage, and that this was not just a consequence of operation but due to the primary pathologic process? I really feel that the significant lesion is in the posterior fossa and that it may very likely be vascular.

Another possibility is an aneurysm, acting as an expanding mass or a tumor, that did not rupture until the time of this surgical procedure. It could have acted as a tumor, expanding and obstructing at the level of the midbrain. Does anyone have any questions or comments?

Doctor Mendelsohn: The report on the ventriculograms was that there was "no evidence of shift, and a slight dilatation that appeared to be symmetrical." Here I think the weight of evidence would be on the findings at ventriculography as to whether there was increased pressure or increased fluid to account for the increased intracranial pressure. You occasionally see tumors in the posterior fossa that don't give evidence of obvious neurologic findings, sometimes, until a patient begins to deteriorate. It's possible to tie in the findings down in the spine, the weakness of the flexors of the foot, the scoliosis, et cetera. In some tumors of the posterior fossa you get seeding of the tumor down the sac and in the meninges, and you can have implants from almost any of the gliomas that could arise in the posterior fossa and seed down there. Likewise, although it would be stretching it, you could tie in the low back findings with bleeding intracranially. Often patients who have subarachnoid hemorrhage complain first, or early in their course, of low back pain, presumably from the blood settling down in the cul-de-sac and irritating the nerve roots. As far as making

a guess, I would say this was a posterior fossa tumor rather than vascular lesion, just on the basis of percentages.

Physician: What would you make of the poor filling of the fourth ventricle?

Doctor Mendelsohn: The lateral ventricles were well filled. As Doctor Baker mentioned, very often for technical reasons you don't fill the fourth ventricle, aqueduct, and even the third ventricle. Where you have clinical evidence that there could be a lesion there, it may be significant. This is especially true when you try to fill it from below and don't. If you don't find evidence of a lesion, you just have to depend on your clinical picture. If there is no reason to expect a lesion there, you just assume it is a technical difficulty. If there is an obstruction at that point, then he should have hydrocephalus. He should have big ventricles. Undoubtedly the surgeon felt that there was hydrocephalus in this case—*i. e.*, that there was increased pressure in the ventricles—otherwise he would not have done a Torkildsen. Presumably he felt there was an obstruction in the ventricular system.

There is one other thing that wasn't mentioned. It is possible for systemic illnesses to cause these same findings. Collagen disease could give the whole picture as far as intracranial and central nervous system findings are concerned.

Doctor Baker: In general, though, one would expect more findings, either systemic or indicating diffuse involvement of the central nervous system. I don't believe it would explain our patient's difficulty.

Doctor Eisenberg:* Couldn't the findings in this case be accounted for by some infectious process such as tuberculous meningitis or a fungus infection?

Doctor Baker: Yes, that is a possibility. We would like very much to have seen a spinal fluid examination and a better general medical evaluation, too.

Dr. Baker's diagnoses:

1. Vascular anomaly or neoplasm at level of midbrain or tentorial incisura, most likely on right side
2. Possible midbrain or cerebellar glioma

Dr. Mendelsohn's diagnosis:

1. Posterior fossa tumor

PATHOLOGIC FINDINGS

Doctor Strong: The last question from the floor was a very pertinent one. The major findings at autopsy were confined to the central nervous system, although there was slight bronchitis and early bronchopneumonia. The brain weighed 1,600 grams, was soft, and held its shape poorly. In the right occipital region the tissue was soft, hemor-

*First Lt. Eugene Eisenberg, USAF (MC), Medical Service.

rhagic, and fragmented. In the left occipital region, a 2-cm defect containing fragmented brain tissue was noted. The area of the hemorrhage noted in the right occipital region extended into the posterior portion of the corpus callosum. Over the convexity of the brain, the sulci showed signs of increased pressure, with swelling and flattening. The vessels were congested. Around the base of the brain there was subarachnoid hemorrhage and softening. Multiple sections through the cerebrum showed the blood in both lateral ventricles and in the third ventricle that had been noted at operation. There was more blood on the right side than on the left. Sections through the mesencephalon showed the aqueduct to be occluded. A fairly large area of hemorrhage was present in the brain stem beneath the aqueduct. Sections from the most cephalic portion of the pons disclosed a normal fourth ventricle. The medulla and cerebellum were not remarkable.

Microscopic examination of sections from the mesencephalon at the level of the inferior and superior colliculus and at the level of the internal capsule showed subarachnoid hemorrhage with necrotic cellular debris present. Among this debris were organisms morphologically identical with *Cryptococcus neoformans* (figs. 1 and 2). These organisms were bluish in color and round to ovoid in shape. There was some suggestion of budding. The organisms had the typical thick capsule

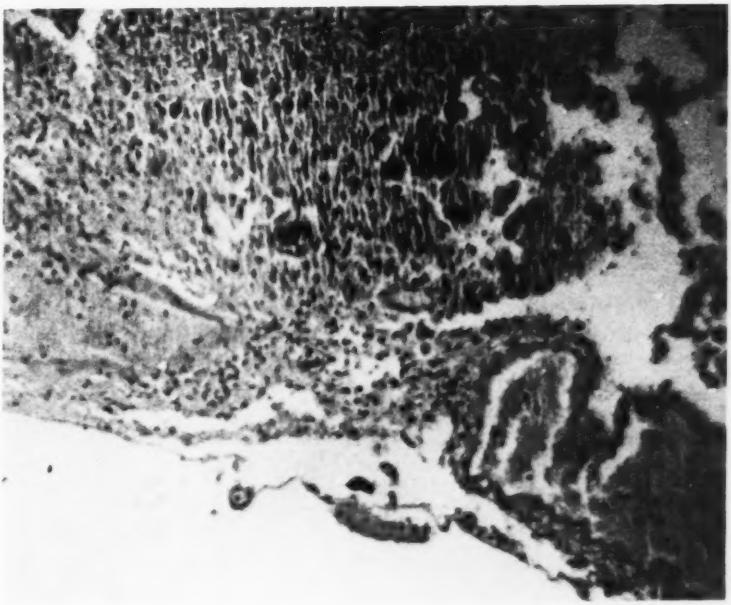

Figure 1. Section showing degeneration and chronic inflammation of brain tissue with Cryptococcus neoformans ***organisms in the area of degeneration. (× 100)***

and ranged in size from 5 to 15 micra in diameter. Also a section from the cerebellar-pontine angle showed organisms both on the surface of the brain and within the brain, being more prominent on the surface.

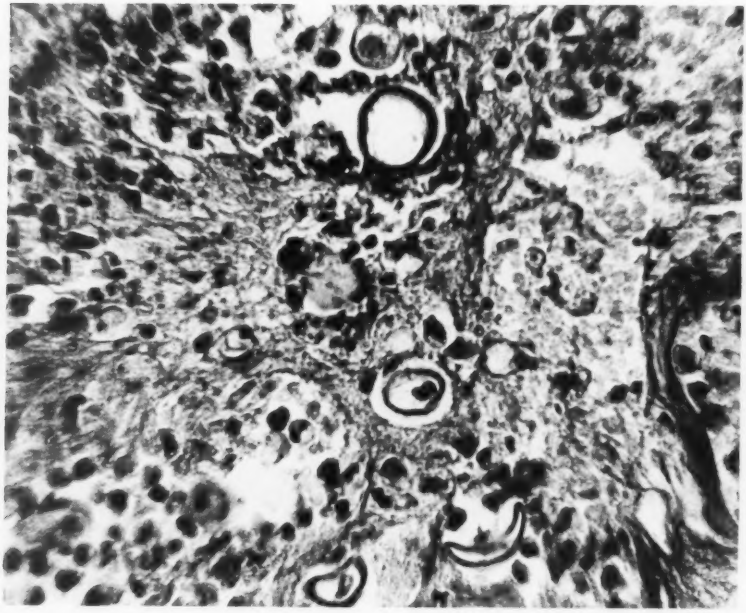

Figure 2. Cryptococcus neoformans *organisms (under high power) in areas of degeneration of brain tissue. (× 400)*

There were no actual gross cysts noted on sectioning but with these findings the diagnosis of cryptococcosis could be made.

Pathologic diagnoses:

1. *Cryptococcus neoformans* meningoencephalitis with obstruction of ventricular system of brain in vicinity of aqueduct of Sylvius
2. Bronchopneumonia, early
3. Focal atelectasis of lung

Doctor Strong: Cryptococcosis is a subacute or chronic infection, caused by a yeast correctly named *C. neoformans* and also known as *Torula histolytica,* which may involve various portions of the body but which has particular predilection for the brain and meninges. According to Ratcliffe and Cook,[1] it has been called European blastomycosis and was first described in Germany by Zenker in 1861. The great majority of cases, however, have occurred in the United States, with only 30 cases recorded from the European continent, so that the term European blastomycosis is a misnomer.[2] This organism has been

found in fruit juices, on the surface of peaches, on plants, in insects,[2] and in the human throat, gastro-intestinal tract, and skin. It has been found in some infections in horses and other animals. There has been no evidence that man has contracted the disease from an infected animal or that the disease is transmitted from man to man. Cryptococci from severe infections are differentiated from those from the above sources only by pathogenicity in animals. This organism grows rather slowly in man, showing that man probably is not a suitable host for the organism.

Cryptococcus grows at room temperature or at 37°C. It forms a moist, slimy growth which is white for the first several days; at the end of approximately one week it begins to turn tan, brown, or orange in color.[3] The organisms are ovoid to round, yeastlike in appearance, and produce single buds. The organisms have thick capsules which are shown best by examining exudate or culture material with dilute India ink. The cryptococci may be differentiated from other budding fungi such as blastomyces by the formation of single buds, their wide capsule, and their failure to produce mycelia on culturing at room temperature.

Clinically, the infection may remain localized or may clear up entirely. If it does continue to spread, it usually spreads by way of the lymphatics and blood stream to various tissues and organs and almost invariably terminates in the central nervous system. When meningitis develops, the course is more rapid and nearly always fatal. In the central nervous system cryptococcosis usually appears gradually, as in this case. Often there are intermittent frontal headaches which later become more severe and continuous. Occasionally the onset is sudden. Dizziness, vertigo, and pain in the back of the neck are common symptoms. As the disease progresses, evidence of severe mental disturbances may occur.[4]

The physical signs are the same as those of any chronic meningitis, with stiffness of neck and positive Kernig and Brudzinski signs. Amblyopia is common and strabismus, nystagmus, ptosis, diplopia, ataxia, and hemiplegia are noted occasionally. Papilledema is frequently present. In spite of the severity of the symptoms, the patients usually do not present the picture of acute infection. There is usually a low-grade fever, slight leukocytosis and relative lymphocytosis, normal blood pressure, and normal or slightly increased pulse rate. As the disease progresses there is marked loss of weight and strength, and the patient ultimately becomes comatose and dies of respiratory failure. Clinically, some of the conditions that cryptococcosis of the central nervous system may simulate are encephalitis, paresis, tuberculous meningitis, brain abscess, subarachnoid hemorrhage, subdural hemorrhage,[5] virus meningitis, pyogenic meningitis, brain tumor, or spinal cord tumor.[6]

Diffuse cryptococcosis, without specific granulomata as we see in this case, can produce the picture of an expanding intracranial lesion and may occasionally do so with a negative spinal fluid. Of

220 patients with cryptococcosis reviewed, 178 had central nervous system involvement; 42 (23 per cent) of these 178 were suspected of having an expanding intracranial or intraspinal lesion and an operation was performed.[2] So the present case is not particularly unusual in that respect. Usually the course of the disease is from one to six months, although there have been cases in which the patient died within several weeks[7] and some in which the patient lived over seven years after the onset. The meninges and the brain may appear practically normal. The organism may be scattered so evenly in the subarachnoid space that cloudiness is scarcely perceptible. The cut surface of the brain may be normal, or there may be tumorlike masses of organisms with apparent cyst formation. In the meninges the organisms are usually abundant in the exudate. In the brain they may occur in the gray matter or basal ganglia and frequently form cystlike areas due to accumulation and coalescence of capsular material.[6] Epithelioid and giant cells, similar to those seen in tuberculosis at times are numerous in the lesions. Other organs or tissues less frequently involved are skin, subcutaneous tissues, lungs, kidneys, liver, spleen, adrenals, and pancreas.

If cryptococcosis and other fungus diseases of the central nervous system are considered in the differential diagnosis both of meningitis and of suspected expanding intracranial lesions, the diagnosis will be made more often during life.[2] The disease may be suspected if there is an unexplained pleocytosis or reduced cerebrospinal fluid sugar, or if there are systemic lesions in presence of neurologic signs and symptoms of undetermined cause. In suspected cases, spinal fluid should be cultured for fungi. Cultures should be incubated for at least one month.[7] One negative culture doesn't exclude the disease. The spinal fluid should be carefully examined using a dilute India ink preparation.

The treatment for this disease is unsatisfactory. Iodides and sulfa drugs have been used with questionable success. It seems likely that the usual progressive course to a fatal termination is best explained by poor antibody response, the protective effect of abundant capsular material, and the inefficient phagocytic reaction of the host's cells.

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WHAT IS PSYCHOANALYSIS

"The thesis that psychoanalysis is a method which helps the individual accept his environment is more than an oversimplification. It is erroneous because it neglects one fundamental aspect of psychoanalytic treatment. Psychoanalysis aims at something more than merely helping the patient adjust himself to the social system in which he lives. Psychoanalysis is a true product of that phase of Western civilization which had deep respect for individual differences that people actually have in our complex free societies. It aids a person in the integrative task in which he failed: to reconcile his own basic personality with his environment without sacrificing that intangible something which makes him a person different from all others. Psychoanalysis is the most individual type of treatment which medical science has ever produced. Each case is a unique problem. What the therapist is primarily interested in is not the nosological classification of a person, not in what way he is similar to others but in what way he differs from them. Every person has his own potential formula of adjustment. Psychoanalysis aims at an adjustment which takes cognizance not only of a given social environment but also of the uniqueness of every person. From this perspective psychoanalysis gives reality to the expression "the respect for the dignity of the individual," a term which is often used in a vague and well-nigh meaningless fashion. This respect for individual difference explains why psychoanalysis is unthinkable and is therefore prohibited in totalitarian societies."

—FRANZ ALEXANDER, M. D.
in *American Journal of Psychiatry*
p. 694, Mar. 1956

MEDICAL PRACTICE WITH THE MARINES ON OCCUPATION DUTY IN KOREA

JAMES B. SHULER, *Captain, MC, USN*

THE NEED for effective medical and surgical support for a combat operation will not be questioned. Few, however, aside from the military personnel actually involved, appreciate the problems of an intact medical section as part of a combat-ready occupation force that may, at any time, be called upon to face renewed hostilities.

With the cessation of actual fighting in Korea in the summer of 1953, the 1st Marine Division, FMF, occupied a frontage on the main line of resistance and was thus obliged to maintain a high order of combat readiness. Historically, this sector was astride one of the main tactical avenues of approach to Seoul from the North. While such an assignment offers excellent training opportunities for the combat units of a division, it could present difficulties in keeping the medical section busy, efficient, and combat-ready professionally.

Surgical and medical skills cannot long remain relatively idle without deterioration. Likewise, hospital corpsmen, ranging from highly trained technicians to junior aid station men, cannot be kept fruitfully busy and professionally fit by simulated situations and combat-training operations alone. The usual medical and surgical conditions met in day-to-day outpatient sick call and inpatient practice, including the ever-present traumatic cases, are not sufficient to keep an adequately staffed medical battalion up to its desired professional capabilities, or to allow for a progressive medical and surgical training program for the medical officers and corpsmen.

NEED FOR MEDICAL AID

The 1st Marine Division's sector of Korea was almost unbelievably devastated from both a social and economic viewpoint. Towns, farms, homes, schools, and people suffered from the horrible destruction and dislocation of war.

As our national policy was orientated toward the rebuilding of Korea, the military forces on the spot were in a position to implement this aim in the place where it was desperately needed.

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The Armed Forces Assistance to Korea Program (AFAK), under the Commanding General, Eighth United States Army, provided funds for rehabilitation projects such as schools, hospitals, aid stations, and local industries, to mention a few important ones in the 1st Marine Division's sector.

For example, under the AFAK program, funds were supplied to purchase the actual building materials for a community hospital. The First Engineer Battalion, as the Division unit sponsor, supplied scientific know-how and supervision in the actual construction of the hospital, which covered the period from the initial plans to the completed structure and official dedication. The local Korean community provided the actual labor for the building phase and the hospital site. The First Medical Battalion acted as professional advisor both for the design of the hospital and for its medical staffing and technical outfitting.

A second higher-level agency concerned with medical rehabilitation and other functions, including public health, was the Korean Civil Assistance Command (KCAC). This organization provided the fitting out of medical and surgical equipment and supplies for the local hospital. It paid the salaries of the local Korean doctors, nurses, and attendants until the hospital was formally transferred to the Ministry of Health. In addition, KCAC supplied the basic Korean ration for the hospital patients. Since this community hospital was in a front line sector and was supervised by the sponsoring First Marine Division units, its growing pains were keenly felt by them (fig. 1).

While the hospital was being constructed, and this took many months, the civilian sick and injured were cared for in a group of temporary buildings adjoining the hospital site. The staff of the hospital consisted of a male and female physician, a dentist, nurses, and attendants—all Koreans.

The Navy medical group, as well as the military in general, were amazed and startled by their first look at medical practice in a native community in which there was so much to do and so little to do it with. Roentgenographic equipment, autoclaves, sterilizers, laboratory facilities, and antibiotics were initially unavailable but not unknown or unappreciated by the local civilian Korean doctors or their patients. With the institution of the AFAK and KCAC programs, the medical supply problem was greatly improved but professional supervision could only be provided by personnel who had something to give and enjoyed giving their services.

MEDICAL PROBLEMS

Many medical officers became enthusiastically interested in local medical and surgical problems. It seemed to take the local populace and local physicians an even shorter time to appreciate

what the Navy doctors had to offer, and there was a huge consultative practice available from the local hospital to the 1st Medical Battalion Headquarters, which was several miles away.

Figure 1. Dedication of Kumchon Community Hospital attended by representatives of the Korean Ministry of Health. The 1st Marine Division Band is in the foreground. Part of the city of Kumchon is seen in the background.

From a public health standpoint, the major medical problem in South Korea was tuberculosis. The extent and degree of tuberculous infection was such that stringent protective measures had to be taken by those of our personnel who saw Korean patients. It did not take long for the skeptic to become convinced that a roentgenogram of the chest was mandatory before any elective operative procedure, no matter how urgent, was undertaken.

Malaria, which was widespread, presented no problem because the military took suppressive therapy during the malarial season. Infestation with intestinal parasites approached the 100 per cent rate among the Koreans.

South Korea, like other parts of the world, suffered its share of the so-called venereal diseases, and gonorrhea was ever present. Syphilis was, to all practical purposes, nonexistent in the 1st Marine Division's sector. We did not diagnose a single case, either by dark field or by serologic means, that was thought to have originated in Korea. Of many hundreds of Kahn tests of civilians performed in the 1st Medical Battalion laboratory, not one was positive. The clinical diagnosis of chancroid could be made fairly often, but laboratory facilities were not available

for confirmation. Some students of this problem in the Far East cast doubt on the actual existence of chancroid in that area.

The local Korean doctors stated they rarely saw cases of hypertensive vascular disease. Toxemias of pregnancy were recognized, but patients so diagnosed by the community hospital doctors were said to have a bad outlook. The general state of nutrition among the natives was excellent, and primary dietary deficiency states apparently were seldom encountered. Pulmonary disease due to *Paragonimus westermani* was frequently diagnosed clinically by the Korean doctors, but, since microscopes and roentgenographic equipment were not at hand, the apparently satisfactory response to treatment (emetine) and the progress of the case differentiated the hemoptysis due to this infection from that due to tuberculosis. One case of pulmonary paragonimiasis in an American military man was diagnosed by finding the ova in the sputum.

The medical problems in Korea were so enormous and of such long-range import that our practice in the civilian population was of necessity limited to the more or less acute medical and surgical conditions, with the exception of corrective and therapeutic procedures in selected cases of chronic diseases and injuries.

For those interested in tuberculosis control measures, however, the tuberculosis control section of the Korean Ministry of Health planned a progressive and widespread program against the disease which will involve the use of large-scale chest photofluorograms, mass tuberculin skin testing of children, BCG vaccine, and current antituberculosis drugs. Most of this latter would of necessity be on an outpatient basis until tuberculosis hospitals can be erected and staffed. It is probable that a part of the facilities of the community hospital in the 1st Marine Division's sector will be used as a sectional photofluorographic, laboratory, and treatment center. This ambitious program, if technical personnel and equipment are obtained, will probably represent one of the most extensive and potentially productive antituberculosis campaigns ever instituted anywhere.

In a similar fashion, opportunities were abundant to get a firsthand look at the tremendously successful infectious and communicable disease control programs of the medical and public health sections of KCAC.

TREATMENT PLAN

With the cessation of hostilities and the local stabilization of the 1st Marine Division units in permanent camp sites, it was not long before the native populace in large numbers were presenting themselves for treatment (fig. 2). In a land where a

physician is a luxury, a Navy doctor with his pain-relieving drugs, equipment, and antibiotics is indeed a wonder. The four operating medical companies of the 1st Medical Battalion, each of which in effect was a small hospital, were, with the exception of medical schools and mission hospitals in the big cities, unique in South Korea. There were few patients indeed in the 1st Marine Division's sector who could ever hope to visit a hospital in a city days away by ox cart or on foot. Thus, in response to obvious need, the medical companies and unit sick bays began taking care of patients whom they were equipped to treat.

Figure 2. Mother and baby who came to one of the more permanent battalion medical installations.

The actual housekeeping procedures incident to the care of inpatients and outpatients presented no special problem. A tent was erected for sick call and to house the patients. Separate outside toilet facilities were set up, the food was provided by KCAC, and the relatives or friends of the patients prepared the Korean ration on Korean stoves and served it. There was never any lack of relatives to do this work, and it was usual for several members of the family to come and stay with the patient. Major operations were done in the medical company operating room.

The 1st Republic of Korea Marine Brigade, later the 1st Republic of Korea Marine Division, was a part of the occupation force in the 1st Marine Division's sector, and there was a United States Marine Advisory Group assigned to the Brigade. A medical officer was a permanent member of this group. A program was instituted by which Korean Navy medical officers and hospital corpsmen were assigned to the 1st Medical Battalion for professional training. One of the principal requirements in selection was the ability to understand English. These medical officers and corpsmen were most anxious to learn, and, where the language barrier did not interfere, they proved to be excellent students. The Korean doctors and corpsmen were given didactic and practical training in operating room technic, laboratory procedures, and patient care. The corpsmen acted as interpreters and ward corpsmen on the Korean civilian wards. The Korean Navy medical officers took care of the civilian Korean patients under supervision and assisted with them in the operating room. The Korean Navy medical officers came to the 1st Medical Battalion on a rotation basis and stayed for three months, then were rotated back to their Marine units and replaced by others..

This program proved to be of great value to both the 1st Marine Division and the 1st Korean Marine Brigade, because it gave our medical people valuable experience in instructing a fighting partner in areas in which we were competent and it gave the Korean medical group a close look at modern American military medicine. At the administrative level, the American Navy doctors could appreciate the various problems and advise their Korean colleagues in matters of organization, supply, and procurement.

SURGICAL PROBLEMS

The orthopedic surgeons had a large experience in the treatment of patients with bone and joint tuberculosis, which was quite prevalent. Previously, these patients had been treated by the so-called "Chinese needle treatment," poultices of various kinds, and incision and open drainage of tuberculous abscesses. Many were rendered completely helpless by bizarre contractures. The patients came to the medical companies by ox cart, A-frame, or astride the back of a relative.

Those patients who would be most likely to respond to surgical treatment were selected. Initially, they were treated on an outpatient basis with streptomycin sulfate, para-aminosalicylic acid, and plaster immobilization. Operations were performed when they were deemed opportune, and the patients were again placed in an outpatient status, usually within two weeks. Follow-ups were necessarily limited, but in general the orthopedist thought the rate of fusion was about the same as in patients in United States hospitals.

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Traumatic conditions were also very prevalent, many resulting from wounds suffered during the fighting, others from stray grenades and mines, still others from the increasing vehicular traffic. An interesting observation by an orthopedist was that in a nine-month period he saw no persons with congenital club foot or hip dysplasia.

In the field of reconstructive and plastic surgery (fig. 3), there were numbers and types of cases to stagger the imagination. A plastic surgeon remarked to the effect that South Korea was the scene of the ultimate in scar contracture. A raw neck, for example, caused the chin to be glued in a scar to the chest; the

Figure 3. Open-air follow-up examinations in the plastic surgery clinic. Inpatient ward tent is in background. Medical officer, corpsman, and interpreter are in left foreground. (Photograph courtesy of Lieutenant D. R. Millard, MC, USNR.)

same process dragged eyelids off the eyes and drew legs up against the thighs. The congenital anomalies of harelip and cleft palate were abundant, but since the native population had not known of the existence of corrective measures, the children would not be brought in. However, once a case had been repaired, it was usual to have others brought in from the same village. As an example, after one particularly successful repair of a harelip and cleft palate in a very young infant who was slowly starving because of inability to swallow, a local papa-san brought three more children with harelip to the medical company on an A-frame.

These procedures were perhaps the most dramatic and impressive long-range results that the Navy medical group performed.

By such means, hopeless cripples were restored to normal activity in their own villages and will stand as tangible evidence of American good will in South Korea for a long time. Since the local hospital physicians and the Korean Navy physicians saw and assisted in many of these repairs, they received valuable experience and practical training.

In the surgical sphere, patients with ruptured peptic ulcers were seen fairly frequently, but often they were not brought in until they were in extremis and suffering from general peritonitis. Patients with appendicitis were frequently seen but likewise at a late stage. An ascaris lumbricoides was sometimes found tightly nested in the appendix. Extensive carcinomas originating in the breast and skin and producing horrible external deformities were common, as were gastro-intestinal cancers.

In the gynecologic field, so-called pelvic inflammatory disease was frequent and responded well to antibiotic therapy. The familiar conditions following childbirth, including relaxed pelvic floor, cystocele, rectocele, and prolapse of the uterus, were thought to be very common, but these patients relatively seldom presented themselves because of ancestral training and their reluctance to appear concerned with things genital. They would often come requesting medicine but would refuse examination. One surgical observer remarked that these childbirth injuries were perhaps one reason why women seldom laugh heartily in this land.

Obstetric situations were very seldom seen. Rarely, a husband would bring in his wife after ineffectual and prolonged labor and in need of operative delivery of a stillborn or macerated fetus. Occasionally a woman would be brought in because the afterbirth could not be passed at home.

Undoubtedly, the most pathetic and inadequately treated patients were those with the severe burns that were so prevalent. One has but to see the crowded conditions in the mud-walled, paper-lined, straw-roofed homes to realize the ever-present danger of fire (fig. 4). For some reason, native Koreans are most reluctant to give blood, and it was seldom possible to give these patients the blood they needed. It was not feasible to start a Korean blood bank in the 1st Medical Battalion laboratory because of lack of donors.

On the more dramatic side, probably the first mitral valvotomy ever performed in South Korea was done in one of the medical companies and with excellent results. Preoperatively a two-hour vehicular trip each way to an Army evacuation hospital for a barium swallow and electrocardiogram was undertaken to confirm the clinical diagnosis of marked mitral stenosis. Another chest

surgeon performed several esophageal operations for stricture incident to accidental ingestion of lye, which is quite common among the children.

Figure 4. Aerial view of a typical small Korean village. This one was just across the fence from the camp site of the 1st Medical Battalion Headquarters. Ricks of unthreshed rice are seen near the houses, the roofs of which are of rice straw.

To illustrate how procedures considered simple by American standards brought the medical group local acclaim is the case of a woman who had been told by her doctor that her enlarging abdomen was due to cancer and thus there was no hope. Examination in a medical company suggested, and operation proved, this to be due to uterine fibroids, but in the minds of the patient and her friends and relatives, she had been cured of cancer. She caused many less fortunate patients to come for treatment.

Lest one think that most of the glory came to the practitioners of the surgical art, the battalion medical officer, with his small outpatient tent but abundant supply of pain-relieving drugs, antibiotics, and scalpels to relieve abscesses, received his full measure in the successful practice of medicine, and the high esteem with which he was held by his grateful patients bordered on awe.

RELATED ADVANTAGES OF PROGRAM

Aside from the humanitarian aspect which was so desperately patent, the training opportunities it afforded the Navy medical

officers, both in keeping them professionally busy and competent and in providing excellent training for our Korean Navy colleagues, this civilian work had its beneficial effect at the unit command level. It paved the way for better co-operation with the local populace through the tightly meshed civilian administrative organization that exists in South Korea. As an example, the Korean community would on occasion petition the local military commander requesting that the doctor be retained when he was being transferred to another assignment. These long and flowery petitions, written laboriously both in Korean and in English, were most complimentary to the individual doctor and to the 1st Marine Division whom he represented.

Again, the Civil Affairs Officer of the 1st Marine Division, through his close association with the civil population, would bring to the personal attention of the Commanding General particularly significant service of medical officers. In a similar manner, the Commanding General of the 1st Korean Marine Brigade would give his personal recognition for the work done by the Navy medical officers in connection with the training of the Korean Navy doctors and corpsmen. The local community hospital gave many individual citations to the doctors who had worked with their patients. Following a series of writeups in the 1st Marine Division's newspaper on this subject, the Pacific edition of *Stars and Stripes* published two feature articles. One told of the general practice of a battalion surgeon, the other was about the plastic and reconstructive operations performed in the 1st Medical Battalion and in the community hospital.

Probably in no place in the world is a greater return available in personal satisfaction for the medical time and effort expended as existed in the war-torn sectors of South Korea. Modern military medicine was truly operating on a frontier of great need.

This practice among the Korean civilians was in addition to the usual acute medical, surgical, and traumatic conditions presenting themselves in a group that exceeded 20,000 personnel. The 1st Medical Battalion was often called on to render medical and surgical care to units from other nearby United Nations Forces, thus affording us the opportunity to observe the customs and the way of life in cultures other than our own. A relatively close association with certain Commonwealth units was mutually very pleasant and educational. The majority of serious medical, surgical, and traumatic conditions that occurred in 1st Korean Marine Brigade military personnel were taken care of in the medical companies of the 1st Medical Battalion.

The rough, bumpy, muddy, or dusty roads in the 1st Marine Division's sector limited vehicular speed to a high average of 15 miles per hour. This, however, interposed no problem in the

transportation of military patients, as full advantage was taken of helicopter evacuation, both in the initial transfer to the medical company and subsequent movement either to the hospital ship or to Army evacuation hospitals when indicated. This rapid, relatively atraumatic method of transportation, particularly of the severely injured, was a time-saving procedure that materially reduced morbidity and sick days and that sometimes was life saving. Medical officers would often remark that they could hospitalize their patients in Korea in a shorter time than they could in the United States.

On the academic side, there were frequent medical meetings to stimulate interest in current and remote professional matters. The 1st Marine Division's medical group had a regular weekly medical meeting. The 1st Medical Battalion had two dinner meetings a week, one a journal club, the other a professional meeting. The 38th Parallel Medical Society of Korea met initially every two weeks and later once monthly. These very fine meetings were often addressed by distinguished visiting physicians and surgeons from the United States. The Military Preventive Medicine Association in Korea met monthly, and its programs were excellent. All of these meetings were from one to two or more hours away by vehicular travel, but it was indeed seldom, if ever, that a medical officer could not find transportation to attend.

Aside from great personal satisfaction which this Korean practice brought to the individual medical officer, most of those eligible found it stimulating and productive, as measured by preparation for specialty board examinations. During one year, four Navy medical officers participated in part one of the American Board of Surgery examinations held in Tokyo. Two others, one an anesthesiologist, another a plastic surgeon, were returned to the United States in a temporary additional duty status to successfully complete their specialty board examinations.

* * *

This brief discussion of medical practice with the 1st Marine Division in Korea has been presented, to outline the opportunities that were available for extensive professional work in the civilian population as well as the military.

LOW-COST ULTRAVIOLET APPARATUS FOR SMALL MEDICAL FACILITIES

ROBERT E. LYONS, *Colonel, USAF (MC)*

IN RECENT years, inexpensive fluorescent sun lamps have been available on the market. These are similar in appearance and electrical characteristics to regular fluorescent lamps, but produce ultraviolet sunburn radiation in approximately the same spectrum as that of natural sunlight. We have found them to be ideal for the construction of a low-cost ultraviolet emitter for use in small hospitals, dispensaries, dermatology clinics, and physicians' offices. The clinical response of dermatology patients treated with this apparatus was satisfactory where an erythema producing secondary tanning was required.

THE LAMP

The fluorescent sun lamp* used in our clinic is a 48-inch tube, coated on the inside with a special phosphor.[1] Radiant energy generated within the tube excites the phosphor coating, causing emission of a large amount of ultraviolet radiation in the spectral range of 2,800 to 3,500 Angström units. The relative radiant energy distribution of the sun lamp as compared with "sunshine" carbon and natural sunlight is schematically represented in figure 1. Ordinates were arbitrarily chosen to put all maximal energy output at the same value. It can be seen that most of the output of the sun lamp occurs at wave lengths between 2,900 and 3,200 Angström units, with a peak at about 3,100 Angström units, whereas relatively little of the energy of "sunshine" carbon or of sunlight lies in this spectral range. The greatest output of "sunshine" carbon actually occurs just below 4,000 Angström units, at the upper part of the ultraviolet spectrum. Practically no radiation below 2,800 Angström units is emitted by the sun lamp, which is an advantage from the standpoint of protection of the patient's eyes.

According to Kovács,[2] maximal erythema effect occurs at 3,000 Angström units, with no capacity to produce erythema above 3,130 Angström units. Blum,[3] on the other hand, gives 2,970 Angström units as the wave length of greatest effect in producing sunburn, with little effect beyond 3,300 Angström units.

From Headquarters, Air Materiel Force, European Area, APO 633, New York, N. Y.

*Westinghouse Fluorescent Sun Lamp FS40T12.

THE APPARATUS

A four-lamp fluorescent light fixture (obtainable as standard equipment at most Air Force bases) was modified by the construction of a polished aluminum reflector backing the lamps and extending, at a 30 degree angle, 8 inches beyond the sides and ends of the fixture. The added reflector, although not an essential feature, appreciably decreased the time required to give a patient an erythema dose of ultraviolet. The apparatus was hung from the ceiling above a table in the treatment room by counterbalanced suspension, thus permitting it to be elevated out of the way when not in use.

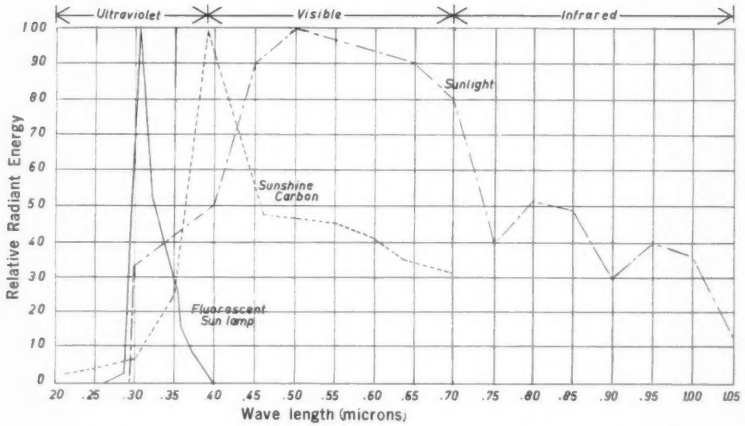

Figure 1. Radiation characteristics (quality) of fluorescent sun lamp, sunshine carbon, and sunlight.

CALIBRATION

Each apparatus should be individually calibrated before being placed in therapeutic use. This is particularly necessary when reflectors are added, because the time in which erythema is produced at a given distance is affected by the type of aluminum used, the angles of the wings, and other variable factors. A standard of calibration is the minimal erythema dose (M.E.D.)—a reaction so slight that it is scarcely noticed by the patient and disappears in less than 24 hours.

Our calibration was accomplished at 20 inches, which we found to be the ideal working distance both for time required to give an erythema dose and for ease of handling the apparatus. In personnel not previously exposed to sunlight or other ultraviolet radiation, 2-inch squares of skin on the inner part of the forearm were exposed for 1 minute; 1 minute, 30 seconds; 2 minutes; and 2 minutes,

30 seconds, respectively. Each patient was examined at 4 hours and again at 24 hours. The square that at the first examination showed a barely perceptible erythema which disappeared in 24 hours, was taken as indicating the minimal erythema dose. The M.E.D. with our apparatus was found to be 1½ minutes at 20 inches, as compared with an M.E.D. of 20 minutes for June sunlight at noon.[4]

RESULTS

The erythema produced by this ultraviolet emitter usually becomes apparent about two hours after exposure. Continued exposure causes a gradual tanning of the skin not unlike that produced by natural sunlight.

We have used the apparatus in psoriasis, atopic dermatitis, pityriasis rosea, alopecia areata, and selected cases of acne vulgaris, as well as for nonspecific tonic effect in other dermatoses. Patients with psoriasis were particularly benefited, and results observed in the other dermatologic conditions were as good as obtained with more expensive types of ultraviolet generators.

Similarity of the fluorescent sun lamp to sunlight in producing sunburn makes it of no value if ultraviolet therapy of the cold quartz type is desired. Emission, as in sunlight, of the 2,587 Angström band is absent.

ADVANTAGES OVER STANDARD EQUIPMENT

Price. The cost of this apparatus, including fixture, aluminum backing, and four sun lamps, should not exceed $50. The lamps have an average life of 2,500 hours when burned at 3 hours per start, and replacements are inexpensive and easily obtained. In an average dermatology clinic using this emitter for all patients requiring it, a lamp change twice a year will ensure maintenance of the standards of emission.

Space Saving. Counterbalanced suspension of this apparatus from the ceiling makes it possible to utilize the same room for other purposes, and avoids loss of floor space such as occurs with many types of ultraviolet generators. This is particularly important where space is at a premium.

Time Saving. Erythemogenic efficiency is many times that of even the strongest sunlight. Also, when indicated, the entire body can be exposed at one treatment.

Minimal Danger to Eyes. The radiation from sun lamps is similar to sunlight in having no component below 2,900 Angström units, so that the likelihood of eye injury from normal exposures is less than with the usual ultraviolet generator. Even so, the patient's eyes should be protected during treatment by special glasses or other means.

SUMMARY

An efficient and inexpensive apparatus for ultraviolet therapy can be constructed at most military bases using commercially available sun lamps in a standard fluorescent light fixture modified by adding an aluminum reflector. The radiation obtained is similar to that of natural sunlight in the erythemogenic range, and clinical response of dermatologic conditions differs in no discernible way from that obtained with natural sunlight. Important advantages include low cost, economy of space, ability to radiate the entire body at one time, and accurate control of dosage.

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"BE PREPARED"

"What have we learned from the great disasters of the past? This may be summed up in a very short phrase: The absolute necessity for proper organization and direction. Without such organization, panic will ensue. This panic may be likened to the stampede of horses and cattle in a burning stable when, by milling about, they eventually destroy themselves rather than accept the way to safety. And so with the ill conceived and almost thoughtless actions of people at such times—mass hysteria if you will, prompt and vigorous leadership may prevent or quell such panic. All of our cities and towns, even our smallest communities, should be ready with a plan of action which includes assistance to their neighbors."

—FRANK B. BERRY, M. D.
in *Bulletin of American College of Surgeons,* p. 66, Mar.-Apr. 1956

Fever of Undetermined Origin

Presumptive Cause, Amebic Colitis

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THE CLINICAL course of amebic colitis is so variable and the causative organism often is so difficult to find, that there is ever-present danger of failure to diagnose a case, with unfortunate results for the patient. He may be asymptomatic, or may complain of any one of a wide variety of symptoms such as constipation, abdominal cramps, nausea, belching, anorexia, malaise, diarrhea, vague aches in the head or extremities, sweating, tachycardia, and fever. Pyrexia was the predominant feature of the following case, in which the evidence supported a presumptive diagnosis of amebiasis.

CASE REPORT

A 29-year-old staff sergeant was admitted to this hospital on 14 February 1955, because of malaise. He had been in good health until in Korea about three months previously he "just did not feel well" and lost his "appetite and energy." He noted backache, slight cough, an occasional sensation of tightness in the chest, and a rare bout of diarrhea that never persisted. No melena was ever noticed. He had lost 35 pounds in the three months. The patient claimed that he ate only government-approved food in Korea and denied drinking unpasteurized milk. Powdered eggs and powdered milk were frequently consumed. He stated that he had taken all prescribed malarial medications as directed.

During the past month, while in transit to the United States, the patient had experienced fever and chills that seemed to occur twice a day. Ten days prior to admission to this hospital, he saw his family physician, who gave him three injections of penicillin without effect on the fever.

There was no history of sore throat, earache, hemoptysis, vomiting, hematemesis, jaundice, or dysuria. The patient had worked in an auto repair shop but had had no contact with any known toxic agents. Although he had been a heavy drinker, he had abstained from alcoholic beverages in the past six months. In 1947 he had been hospitalized

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because of an acute epididymitis and treated with "120 penicillin injections." Further than this, the history was noncontributory.

Physical examination on admission revealed a patient who appeared cachectic and was acutely ill. He had moist skin, a pulse rate of 92 per minute, and a tempterature of 102°F by mouth. Otherwise, the examination was essentially negative, except that he complained of mild tenderness on palpation in the right lower part of the abdomen.

Initial laboratory data included a hemoglobin level of 11.8 g per 100 ml and a white blood cell count of 10,150 per μl, with a differential of 58 per cent polymorphonucleocytes, 36 per cent lymphocytes, 2 per cent monocytes, 3 per cent eosinophils, and 1 per cent basophils. The corrected sedimentation rate was 30 mm/hr. The specific gravity of the urine was 1.010, and microscopic examination of the sediment revealed from 10 to 12 white blood cells per high power field. Roentgenograms of the chest were within normal limits.

Further laboratory reports showed a nonprotein nitrogen of 45 mg per 100 ml; total protein, 9.7 g per 100 ml; albumin, 6.3 g per 100 ml; globulin, 3.4 g per 100 ml; icteric index. 3.5 units; total serum bilirubin level, 0.3 mg per 100 ml; alkaline phosphatase level, 1.5 Bodansky units. The sulfobromophthalein sodium test showed 2 per cent retention of dye in 45 minutes. The heterophil test revealed a positive titer at 1:14. A 12-lead electrocardiogram showed no axis deviation; sinus tachycardia, flat T waves in aVL. The P-R interval was 0.15 second. Three gastric analyses were performed. The specimens revealed no acid-fact organisms on smear or culture. On two of the gastric specimens no free hydrochloric acid was detected. (A histamine test was not given.) Second strength PPD skin test was positive.

For the first three weeks of hospitalization the patient ran a septic course with temperatures varying between 99°F and 103°F. He was placed on the seriously ill list and continued to lose weight although supplementary feedings were encouraged. No bacterial growth was noted from blood cultures made on six separate days when the temperature was over 101°F. Cultures also were made of two fecal and two urine specimens, but no bacterial pathogens were found. Five blood smears, both thick and thin, made when the temperature was elevated, revealed no malarial parasites. All fecal specimens, including enema products, were taken directly to the laboratory, and 20 examinations were made for parasites and ova, with negative results. An occasional stool guaiac test showed a trace reaction, but the patient had not been on a meat-free diet. Febrile agglutination studies were performed for brucella, typhosa "O", typhosa "H", paratyphoid "A", paratyphoid "B", OXK, OX2, and OX19. Titers were repeated after a 14-day interval, and no diagnostic abnormalities were noted. Several repeat complete blood cell counts revealed an occasional mild eosinophilia with white blood cell counts varying from 8 to 12,000 per μl. The sedimentation rate varied between 30 and 40 mm/hr. Several urine sediments showed 8 to 10 white blood cells per high power field. Although an intravenous

pyelogram revealed a small retention cyst in the right kidney, the psoas shadows were normal, no significant abnormality was encountered, and repeat physical examinations showed no change.

A proctoscopy performed under caudal anesthesia because the patient developed acutely inflamed, painful, edematous external hemorrhoids, revealed a normal mucosa to 10 cm, and smears of the mucosa showed no parasites or ova. A barium enema, given after the proctoscopy, passed unobstructed to the cecum. In the cecal area the mucosa appeared serrated, with a thickened, irregular border (fig. 1).

Figure 1. Roentgenogram taken after barium enema. The mucosa of the cecal area appears serrated, with a thickened, irregular border.

In view of fluoroscopic and roentgenologic findings that were suggestive of an inflammatory colitis in the cecum, and of the patient's febrile illness and recent return from Korea, therapeutic trial of an amebacide was warranted. Chiniofon, in 0.75-gram doses three times a day for 10 days, produced a striking clinical response. In 48 hours the temperature dropped to 98°F and stayed below 99°F for the remaining 20 days of hospitalization. The patient's toxic appearance rapidly

disappeared; he felt entirely well, had a healthy appetite, and proceeded to gain 10 pounds.

Because the patient had no free hydrochloric acid in his stomach contents on two gastric analyses, an upper gastro-intestinal study was performed prior to discharge from the hospital. No intrinsic disease of the esophagus or stomach was demonstrated. The duodenal bulb was normal, and films taken at 1-, 4-, 8-, and 24-hour intervals revealed a normal small bowel pattern. Several pictures taken as the barium filled the cecal area from above showed that the inflammatory changes which had been noted with the earlier barium enema were no longer present.

After 37 days of hospitalization, the patient was returned to active duty. Multivitamin tablets were prescribed and he was followed in the medical clinic. Physical improvement was rapid, he regained 25 pounds, and a repeat barium enema, eight weeks after discharge from the hospital, showed the cecum to be normal.

DISCUSSION

In the differential diagnosis of prolonged fevers of obscure origin, amebiasis should be given more serious consideration than it usually is accorded. Demonstration of trophozoites or cysts of *Entameba histolytica* may be difficult where no significant diarrhea is present, but when a thorough investigation of the cause of an obscure fever or prolonged diarrhea is unsuccessful, a therapeutic trial of an effective amebacide may bring gratifying results,[1] provided the choice of drug or combination of drugs is suited to the sites of localization.[2,3] In the case presented, neither trophozoites nor cysts could be found in spite of the most careful search; however, there was a dramatic response to chiniofon that strongly supports a presumptive diagnosis of amebiasis.

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THE RESERVE OFFICER

A highly trained, well-organized and powerful reserve force in constant readiness is vital to the survival of our way of life. With its valuable experience and its faithful regard for country the Reserve Officers Association is representative of a significant goal of our reserve program—the trained reserve officer.

—MATTHEW B. RIDGWAY, General, USA

A True Hermaphrodite

EVAN L. LEWIS, *Major, MC, USA*
RICHARD W. CLETSOWAY, *Captain, MC, USA*

IN SEVERAL recent articles,[1,2] reviews of the literature have been made, bringing the total number of true hermaphrodites reported to 56 as of the date of writing. Due to the length of time between writing and publishing an article, there is a possibility of others being reported, but it is assumed that this is the 57th published case of a true hermaphrodite.

CASE REPORT

A 10-year-old "boy" was brought to the urological clinic at this hospital for repair of hypospadias with chordee and orchiopexies. At birth it was noted that the testes were absent and there was a perineal hypospadias. His development was otherwise normal. At the age of 2 years he was taken to a urologist who advised delay of any surgical intervention until 10 years of age. At 6 years of age, a short course of gonadotropins was given without results. His general growth pattern and activities were normal for a boy of this age.

Physical examination on admission revealed a normal child except for the genitalia. The penis was of normal size but there was a marked chordee with bifid scrotum and a perineal hypospadias. The scrotal sacs were empty, and there was no evidence of a hernia. Rectal examination failed to reveal any evidence of a prostate. On 15 March 1955, under general anesthesia, cystoscopic examination revealed a dimple in the posterior urethra 0.5 cm in diameter and approximately 1.5 cm deep. The tip of the cystoscope was inserted, but no cervix or other opening could be observed. The bladder was normal. There was no prostatic urethra and no colliculus seminalis (verumontanum).

A midline incision was made in the abdomen. Examination of the pelvis revealed what appeared to be a testis, measuring 2 by 1 by 1 cm, on the right side. This organ was connected to a tube which coursed the pelvic wall and the posterior vesical surface to the midline. The testis was entirely intraperitoneal. On the left side there was a tube with a fimbriated end. Enmeshed in the fimbria was an organ 10 by 5 by 3 millimeters. The tube went along the pelvic wall and joined with the right one on the posterior wall of the bladder. There was no evidence of a uterus. The left gonad and tube were removed completely, including the point of junction with the tube from the right side. The

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right gonad was biopsied. An appendectomy and removal of a large Meckel's diverticulum were done as incidental procedures. A first stage hypospadias repair was done. Recovery was uneventful.

Pathologist's* Report:

The section of testicle, measuring 0.8 by 0.5 cm (fig. 1), contained about 16 lobuli testis, each of which contained about 100 seminiferous tubules measuring 40 microns on cross section. Some of the tubules were elongated, measuring as much as 100 microns in length. The

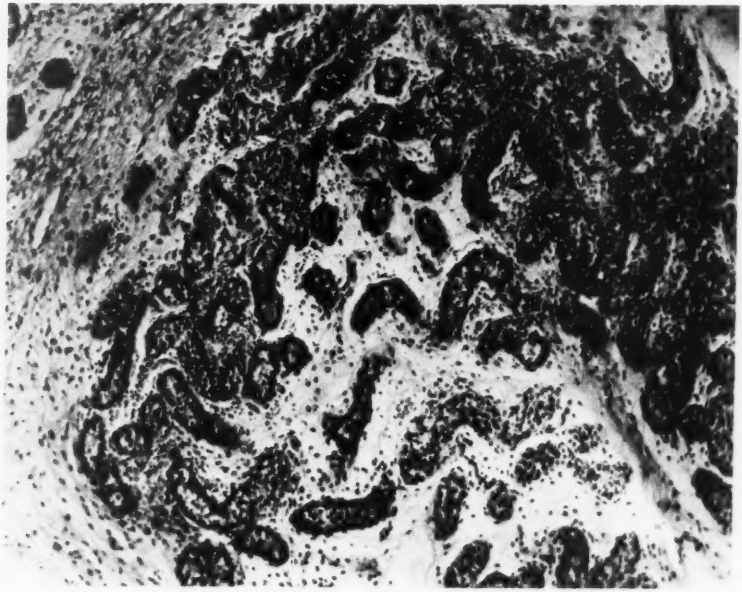

Figure 1. Biopsy specimen of testis showing immature tubules and lack of interstitial cells. (× 110)

tubules were lined by single cells which formed a syncytium. In some instances, the lumens contained large cells measuring 10 to 16 microns in diameter and resembling spermatogonia. Other stages of spermatogenesis were not present. The lumens in some instances contained acidophilic staining material. The interstitial tissue contained blood vessels, nerves, and connective tissue. The connective tissue had an edematous appearance. No interstitial cells were present. The periphery of the testicle was composed of thickened tunica measuring as much as 60 microns in diameter. In and adjacent to the tunica, there were expanses of tubules which were arranged in a lobular pattern.

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*Major M. David Orrahood, MC, USAR

Sections of ovary and fallopian tube were present on the same slide. The ovary measured 5 by 3 mm in size and was lined by a flattened layer of endothelial cells (fig. 2). The medulla as well as the cortex of the ovary contained about 24 follicles measuring 40 to 70 microns in diameter. The cysts were lined by a single layer of columnar cells which formed small cumuli in some areas. The follicles were surrounded by a ring of compressed stroma cells forming theca externa. The follicles contained varying amounts of acidophilic staining material. The stroma of the ovary was composed of a loose network of fibrous tissue

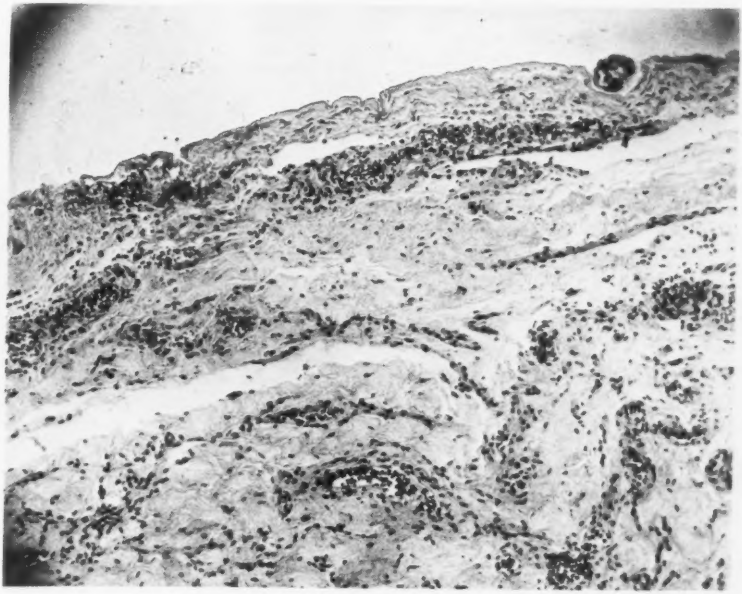

Figure 2. Biopsy specimen of immature ovary. (× 110)

with few cells. No hilar cells or characteristic stroma of adult ovary were observed. Several areas of stroma contained recent hemorrhage.

The fallopian tube was well formed and free of abnormality. The tube was approximately 5 mm in diameter (fig. 3).

Comment

This patient had been reared as a boy and although at the time of surgical exploration it would have been easy to have changed his sex, we decided not to. We are in agreement with the previously expressed opinion that the psychic trauma is too great if the sex is changed after 3 or 4 years of age. Reproduction was not a factor in either case, and a copulatory organ is a matter of plastic surgery, male or female. The testis could not be

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brought into the scrotum so plastic prostheses will be considered at a later date.

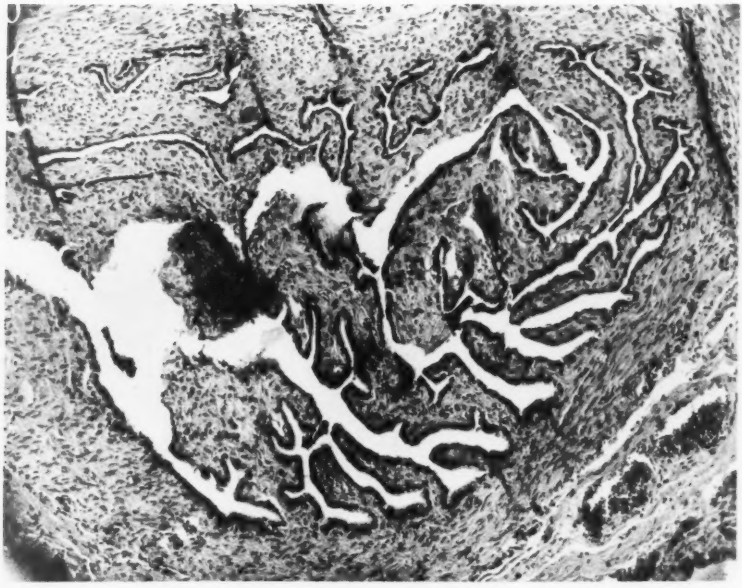

Figure 3. Section of fimbriated end of fallopian tube. (× 110)

The vaginal dimple was not disturbed since experience has shown that its removal is not necessary and is, in fact, contraindicated.

The biggest problem in handling this patient will be at puberty. It is anticipated that male hormone therapy will be necessary due to the absence of interstitial Leydig cells.

There are several interesting and unusual features in this patient. Both gonads were extremely immature. The tubular structure from the left gonad represents the müllerian ducts, and the one from the right definitely appeared of wolffian origin. The point of fusion was removed but had no histologic resemblance to a uterus and was not connected to the vaginal dimple. The arrest of development at such an early stage is unusual. That neither a prostate nor a uterus formed, that the vagina is small, and that both gonads are extremely immature bears out this fact. We believe that this patient is probably a gynandromorph—at most, only one stage further developed than a neuter. There are only two patients in the literature showing anywhere near this immaturity: that of Schapiro[3] and that of Davis and Scheffey.[4]

The latter was made female at the age of 6 years, due to a good vagina and a small phallus.

SUMMARY

The 57th case of a true hermaphrodite is presented. This patient probably represents a gynandromorph with extremely early arrest of development and comes under the classification of hermaphroditismus verus lateralis.[5] The characteristic features are: right testis and vas, left ovary and tube, absent uterus, very small vagina opening into posterior urethra, absent prostate, hypospadic phallus with chordee, cleft scrotum, and perineal urethra.

ADDENDUM: Since this article was submitted, a skin biopsy was made (in January 1956) and revealed a predominance of male chromosomes in the nuclei of the basal cells.

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THE MARK OF THE PHYSICIAN

"The mark of the true physician is his sensitivity to the needs and suffering of others. Perhaps compassion is the word best fitted to express the feeling of a physician for his suffering patient, but this does not mean that the physician can lose his objectivity while being sympathetic with his patient. Neither does it mean that his defense against his own anxiety and concern for the patient should take the form of cynicism, or a coldly scientific, hard-bitten exterior manner."

—DANA L. FARNSWORTH, M. D.
in *New England Journal of Medicine*, pp. 561-562, Mar. 22, 1956

TRIAL OF NEW RESPIRATORY VACCINE SUCCESSFUL

A new vaccine, developed and prepared by the Department of Respiratory Diseases at the Walter Reed Army Institute of Research, Washington, D. C., has been found to reduce the incidence of hospitalization for respiratory disease among recruits by more than 80 percent, according to a preliminary report to the Surgeon General, Department of the Army.

The vaccine was prepared from tissue cultures of monkey kidney which had been infected with the two predominant RI (also called ARD and APC) viruses. Virus in the vaccine was killed with formaldehyde, and the vaccine caused no untoward effects in the more than 350 persons who received it. The new vaccine reaches its maximum effectiveness within a week after administration.

MICROBIAL IDENTIFICATION AID

A handy microbial identification wheel, devised by Colonel H. A. Van Auken, MC, USA, and Major C. D. Graber, MSC, USA, of the Laboratory Service, Fitzsimons Army Hospital, Denver, Colo., is available without cost from either the Difco Laboratories, Detroit 1, Mich., or Parke, Davis & Company, Detroit 32, Mich.

Various biochemical and serologic characteristics of an organism are given by turning the inside wheel until its arrow points to the suspected organism on the outer wheel.

A MESSAGE FROM THE A. M. A.

Since January and in each successive issue of the *United States Armed Forces Medical Journal,* the A. M. A. message has been devoted to the history, organization, function, and purpose of the American Medical Association. The message this month concerns the Council on Medical Physics.

The American Medical Association House of Delegates, in December 1955 at Boston, approved a recommendation to reorganize the Council on Physical Medicine and Rehabilitation and to rename it the Council on Medical Physics. The Council on Physical Medicine and Rehabilitation, formerly the Council on Physical Therapy, was established in 1925. At that time physical medicine was a controversial field. Some physicians believed it had a great future, and others regarded it with misgivings. The former Council helped to bring order to this field and through evaluation of devices and methods it encouraged further development and utilization of many types of instruments throughout all branches of medicine.

The new Council on Medical Physics, one of the nine standing committees of the Board of Trustees, will carry on some of the functions of the former Council together with other new activities as a part of its program. In the reorganization plan the House of Delegates indicated that rehabilitation problems should be assigned to an intra-association liaison group with representatives from all interested American Medical Association councils, sections, and committees dealing with the subject.

The House of Delegates and the Board of Trustees have directed the Council on Medical Physics to carry on activities in the fields of medical apparatus, instruments, and devices. The membership of the Council will include physicians and other scientists with knowledge in the basic and medical science fields of internal medicine, physical medicine, orthopedic surgery, biophysics, radiology, physiology, otology, experimental medicine and surgery, ophthalmology, anesthesiology, pathology, dermatology, and neurology.

The purpose of the Council is in keeping with the service program of the American Medical Association to the medical profession and the public. The purpose might be stated to be the encouragement of an understanding of the actions and rational

From the Council on National Defense of the American Medical Association. The views and opinions expressed are not necessarily those of the Department of Defense. —*Editor*

uses of apparatus and methods having a physical basis for their actions for the diagnosis, treatment, and prevention of disease.

In the pursuit of this purpose, it is expected the Council will conduct a program for the evaluation of new apparatus and physical methods intended for diagnostic, therapeutic, or preventive purposes. Also, the Council may be expected to encourage the development of standards for medical apparatus by appropriate standardizing agencies. The encouragement of research to help establish the actions and rational uses of new classes of apparatus, instruments, and devices is expected to be an important challenge to this newly established Council. The Council's operation will not include the consideration of individual brands and models of devices but rather will be directed toward reporting to the profession on various classes of apparatus.

For example, an individual resuscitator would not be tested and evaluated and as such be the subject of a Council report. Instead the Council, following an extensive project of general analysis and evaluation, would consider the preparation of a report on the desirable features, actions, uses, and contra-indications for manual and mechanical methods of artificial respiration. Generally, reports of the Council would contain information regarding the actions, uses, dosage, contra-indications, and warnings regarding limits of safety for the class of apparatus under consideration.

When one reflects on the almost staggering increase in the use of instrumentation in both laboratory and clinical medicine for therapeutic, diagnostic, preventive, and research purposes, it becomes apparent that the Council can serve all of medicine by encouraging their rational use and understanding of the basic principles of physics utilized in the world of medicine today.

The organizational meeting of the Council on Medical Physics is scheduled for mid-June. Shortly after that, the Council will publish an article in *The Journal of the American Medical Association* on its new program. Quite obviously, the work of the Council cannot progress smoothly nor can its goal be accomplished without the advice and help of many physicians and other scientists throughout the country whom the Council will call upon for their expert opinion.

The research and development programs in the medical research laboratories and clinics of the Armed Forces have made significant contributions in the application of technics for the medical care of service personnel and civilians alike. The availability of such reports from these excellent military institutions should prove of immeasurable benefit to the Council in the conduct of its work. The Council anticipates harmonious relationships with the military representatives of these laboratories and clinics.

Promotions of Officers

The following officers of the military medical services on active duty in the Army, Navy, and Air Force have recently received temporary promotions to the rank indicated.

MEDICAL CORPS

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ALEXANDER, John E., Capt., USAF
ALEXANDER, Richmond L., Jr., Capt., USAF
ALT, William J., Capt., USAF
ANDERSON, David G., Capt., USAF
ANDERSON, Paul B., Capt., USAF
ANDREWS, Hugh K., Lt., USN
ANTELIS, Eugene, Capt., USAF
AUER, George G., Capt., USAF
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BAITCH, Arthur, Capt., USAF
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BARBE, Robert F., Capt., USAF
BARKER, Walter L., Lt., USN
BARNES, Shelby D., Capt., USAF
BARROWS, Howard S., Capt., USAF
BATES, James S., Capt., USAF
BATSON, Andrew P., Capt., USAF
BAYLEY, John F., Jr., Lt., USN
BAZIL, Gilbert M., Lt., USN
BEACH, Norman F., Capt., USAF
BEAVAN, Reginald A., Jr., Capt., USAF
BEAVERS, William R., Capt., USAF
BENDERSKY, Gordon, Capt., USAF
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BERMAN, Harold J., Capt., USAF
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BOXCO, Julius A. S., Capt., USAF
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BOYLE, James J., Jr., Capt., USAF
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BRYK, David P., Lt., USN
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BURGOS, William F., Lt., USN
BURTON, Robert D., Capt., USAF
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CARPENTER, Albert A., Lt., USN
CARPENTER, John R., Capt., USAF
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CHANEY, James W., Lt., USN
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CHUNN, Samuel P., Capt., USAF
CLARK, Orville S., Capt., USAF
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COFFEY, William F. X., Lt., USN
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ELDREDGE, James H., Lt., USN
ENRIQUEZ, Jorge C., Lt., USN
ENSLEN, Philip J., Lt., USN
ERVANIAN, Alexander, Lt., USN
EVANS, Clarence C., Capt., USAF
EVENSON, David J., Capt., USAF
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GAUER, Robert J., Capt., USAF
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GROW, Buel K., Jr., Capt., USAF
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KINGERY, Frederick J., Capt., USAF
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KLASS, Donald W., Capt., USAF
KORNFELD, Donald S., Capt., USAF
KOTT, Alexander, Capt., USAF
KRAMER, Charles J., Capt., USAF
KRAMER, Norman C., Capt., USAF
KRAUSE, Arnold N., Capt., USAF
KRAVETZ, Russell S., Lt., USN
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ROSENGARTEN, Robert, Capt., USAF
ROTCHFORD, James P., Capt., USAF
ROUSE, Robert M., Capt., USAF

RUCCI, Eustine P., Lt., USN
RUIZ, Frederick J., Sr., Capt., USAF
RUTTEN, Ruben J., Lt., USN
RYON, Alden B., Capt., USAF
SAADE, John E., Capt., USAF
SAMET, Charles M., Capt., USAF
SANDBERG, Hershel, Lt., USN
SATTER, Erby J., Capt., USAF
SAUNDERS, John C., Capt., USAF
SAUNDERS, Stephen B., Lt., USN
SAVUKINAS, Vincent E., Capt., USAF
SCANDURA, Rosario A., Lt., USN
SCHAUB, David H., Capt., USAF
SCHEFSTAD, Wilbur J., Lt., USN
SCHIRACK, John D., Capt., USAF
SCHNUTE, Richard B., Lt., USN
SCHONEBERG, Sheldon, Capt., USAF
SCHULTZ, David W., Lt., USN
SCHULZE, Victor E., Capt., USAF
SEARS, Marvin L., Capt., USAF
SEAY, James E., Capt., USAF
SELANDER, Claf E., Capt., USAF
SELINGER, Harold, Capt., USAF
SHANE, Morton, Capt., USAF
SHARPE, Eugene B., Capt., USAF
SHROPULOS, George P., Capt., USAF
SHUGOLL, Gerald L., Lt., USN
SHULMAN, Kenneth, Lt., USN
SINGER, Ellis P., Capt., USAF
SMITH, Clarence G., Capt., USAF
SMITH, Claude A., Capt., USAF
SMITH, Marion L., Lt., USN
SMITH, Thomas H., Jr., Lt., USN
SMITH, Wallace B., Lt., USN
SMYTH, James W., Lt., USN
SPEAR, Horace L., Capt., USAF
SPECKMAN, Glenn R., Lt., USN
SPIERLING, Paul R., Jr., Lt., USN
STADER, Richard O., Lt., USN
STAMM, Charles F., Capt., USAF
STEMMER, Edward A., Capt., USAF
STENZEL, David M., Lt., USN
STEPH, Donal W., Capt., USAF
STEWARD, John P., Lt., USN
STICKEL, Delford L., Lt., USN
STRICKLAND, William H., Capt., USAF
SUER, William D., Lt., USN
SULLIVAN, James J., Lt., USN
SWIHART, Danny D., Capt., USAF
TAYLOR, Harry C., Capt., USAF
TAYLOR, Robert W., Lt., USN
TOBIN, John A., Lt., USN
TRETTEL, Raymond J., Lt., USN
TRUMPOUR, Donald J., Capt., USAF
TSCHETTER, Clifford N., Capt., USAF
TUPPER, James W., Lt., USN
TURKEWITZ, Hyman, Capt., USAF
UETZMANN, Immanuel F., Capt., USAF
UNGER, Pat B., Lt., USN
UTHLAUT, William W., Lt., USN
VALASKE, Martin J., Lt., USN
VASQUEZ, Mario A., Lt., USN
VASSALLO, Michael, Lt., USN
VISCARDI, John P., Jr., Capt., USAF
VOSS, Marlyn W., Lt., USN
WALL, Thomas, Capt., USAF
WALLACE, Edward R., Lt., USN
WALMSLEY, George L., Capt., USAF
WALTER, William J., Lt., USN
WARMUND, Stanley H., Lt., USN
WARNER, Richard R. P., Capt., USAF
WARREN, James C., Lt., USN
WATERMANN, Clarence E., Lt., USN
WATT, Russell H., Jr., Capt., USAF
WAYS, Peter C., Capt., USAF
WEBB, Edward D., Capt., USAF
WEBER, Murry K., Lt., USN
WEINER, Allan D., Lt., USN
WEINER, Bernard K., Capt., USAF
WEINER, Israel H., Capt., USAF
WEISS, Albert E., Lt., USN
WELZANT, Walter R., Lt., USN
WERR, Joseph A., Lt., USN
WEYMANN, Donald R., Capt., USAF
WHEELER, Jewitt E., Jr., Capt., USAF
WHITLOCK, Charles B., Lt., USN
WICKHAM, Robert A., Capt., USAF
WILLIAMS, Jack B., Capt., USAF
WILLIAMS, Thomas W., Lt., USN
WILSON, James D., Capt., USAF
WILSON, Robert J., Lt., USN
WING, John E., Jr., Lt., USN
WINOGRAD, Herbert L., Capt., USAF
WISE, John D., Capt., USAF
WISEMAN, James A., Lt., USN
WOLANSKY, Jacob, Lt., USN
WORSHAM, Julius B., Jr., Lt., USN
YARBERRY, Otha H., Jr., Lt., USN
YOUNG, William F., Capt., USAF
YOW, John S., Jr., Capt., USAF
YUNGHANS, Carl, Lt., USN
ZARTMAN, Harvey F., Capt., USAF
ZIMMERMAN, Marvin P., Capt., USAF
ZUCKERMAN, Marvin B., Capt., USAF

DENTAL CORPS

ACKEL, Fred J., Capt., USAF
ARCHAMBAULT, Jean B., Capt., USAF
AUSTIN, John A., Capt., USAF
BANGE, Albert A., Capt., USAF
BARTON, John A., Jr., Capt., USAF
BENGTSON, Bengt C. S., Capt., USAF
BERGER, Gerald E., Capt., USAF
BLACKWOOD, Robert C., Capt., USAF
BOTT, Claire J., Capt., USAF
BOYD, Robert N., III, Capt., USAF
BRANSTETTER, Billy D., Capt., USAF
BRANTFORD, Myron W., Capt., USAF
CALDERON, Herbert, Capt., USAF
CARNOW, Ralph, Capt., USAF
CARTWRIGHT, Dale J., Capt., USAF
CHAPMAN, Morgan R., Jr., Capt., USAF
CHRISTENSEN, Duayne D., Capt., USAF
CONNELLY, Cecil C., Jr., Capt., USAF
COOLE, William F., Capt., USAF
CORNYN, John, Capt., USAF
CUNNINGHAM, Joseph B., Capt., USAF
CUSH, Joseph W., Capt., USAF
DANNER, Clark D., Capt., USAF
DEAN, Lucius L., Jr., Capt., USAF
DICKINSON, C. E., III, Lt., USN
DIENER, Allan L., Capt., USAF
DOLCE, Robert G., Capt., USAF
DOLIVEIRA, Julio V., Lt., USN
DORAN, Thomas T., Lt., USN
DREWITZ, James C., Lt., USN
DUFFY, Paul E., Lt., USN
DUGAL, George L., Capt., USAF
DUNWOODY, William J., Jr., Lt., USN
DWORETZKY, Philip J., Capt., USAF
EADES, Prince A., Jr., Capt., USAF
EISENBERG, Myron D., Lt., USN
ELMAJIAN, Kersam E., Lt., USN
ENGEL, Harold J., Lt., USN
FINKELSTEIN, Gerald A., Capt., USAF
FOLTZ, William M., Capt., USAF
FONTENOT, Wilfred J., Capt., USAF
FRIEDMAN, Bernard K., Capt., USAF
FURR, Robert E., Capt., USAF
GERSUK, Jack B., Capt., USAF

GOLDSTEIN, Herbert A., Lt., USN
GONZALES, Roy R., Capt., USAF
GOODSETT, David M., Jr., Capt., USAF
GORDON, Edward R., Lt., USN
GORDON, Herbert P., Capt., USAF
GORDON, William, Lt., USN
GORMAN, Walter J., Lt., USN
GREEK, William J., Lt., USN
GREEN, John M., Capt., USAF
GREENLEE, Richard F., Lt., USN
GREMBAN, Harry A., Lt., USN
GROVE, Raymond L., Capt., USAF
GRUNDMEYER, Elmer P., Jr., Capt., USAF
HALE, William R., Capt., USAF
HALL, Arthur E., Capt., USAF
HAMULA, Warren, Capt., USAF
HATION, Edward I., Capt., USAF
HAYES, David L., Capt., USAF
HEATH, Robert W., Capt., USAF
HEILBRUNN, Fred D., Capt., USAF
HEJL, Anton J., Jr., Capt., USAF
HENNY, Frank A., Capt., USAF
HIRSCHMAN, Lewis A., Lt., USN
HOGAN, David W., Capt., USAF
HOLMES, John B., Lt., USN
HOROWITZ, Jerome E., Lt., USN
HUBER, George B., Lt., USN
HUDSON, Billy B., Capt., USAF
HULSEBOSCH, Thomas A., Capt., USAF
HUSCHART, Vincent J., Capt., USAF
HYNES, Francis J., Lt., USN
IIDA, Harry T., Capt., USAF
JENNINGS, Edward J., Jr., Lt., USN
JOHNSON, Ardee, Capt., USAF
KELLY, Thomas W., Capt., USAF
KOEHLER, Gilbert L., Capt., USAF
KORN, Sheldon, Capt., USAF
KRATOCHWILL, Michael J., Capt., USAF
KRZWICKI, Stanley A., Capt., USAF
LAIKS, Lawrence G., Capt., USAF
LANEY, William R., Capt., USAF
LAWSON, James D., Capt., USAF
LEGAULT, Robert L., Capt., USAF
LEIFER, Calvin, Capt., USAF
LEUIN, Irving S., Capt., USAF
LIPPI, Anthony A., Capt., USAF
LOPEZ, Armando G., Capt., USAF
LURIE, Bertram S., Capt., USAF
MAASS, Robert J., Capt., USAF
MAEURER, John E., Capt., USAF
MAGUIRE, Hugh F., Capt., USAF
MASON, Max G., Capt., USAF
MASTY, James A., Capt., USAF
McLAUGHLIN, Dennis L. J., Lt., USN
McLAUGHLIN, Wayne L., Capt., USAF
MELLANG, Maynard A., Capt., USAF
MELLERT, William D., Lt., USN
MERCER, James F., Capt., USAF
METZGER, Francis G., Capt., USAF
MEULLER, Herbert J., Lt., USN
MILLER, Jack L., Lt., USN
MILLER, John H., Capt., USAF
MILLICAN, Richard D., Capt., USAF
MILLIETTE, Thomas R., Lt., USN
MITCHELL, Gerald A., Jr., Capt., USAF
MOLL, Richard A., Lt. Col., USA
MONTGOMERY, Ralph E., Capt., USAF
MOORE, Andrew, Capt., USAF
MORAY, Robert S., Capt., USAF
MORRIS, Robert W., Lt., USN
MOYEL, Robert B., Capt., USAF
NELMS, Tip C., Capt., USAF
ORTELERE, Robert A., Capt., USAF
OWENS, John J., Capt., USAF
PARADIS, Renald A., Lt., USN
PARKER, Donald R., Lt., USN
PARKER, Leroy A., Lt., USN
PARKES, Robert B., Capt., USAF
PAVLIKOWSKI, Fred L., Capt., USAF
PEKIN, Richard J., Lt., USN
PELTIER, James R., Capt., USAF
PENNY, William G., Capt., USAF
PERKINS, Robert H., Capt., USAF
PETERSEN, Sidney D., Jr., Capt., USAF
PETLAK, Frank R., Jr., Capt., USAF
PIKE, Frederick P., Capt., USAF
PON, Michael F. M., Capt., USAF
POWERS, George E., Capt., USAF
REYNOLDS, Dean R., Capt., USAF
REYNOLDS, Thomas E., Capt., USAF
ROLLINS, Robert W., Capt., USAF
ROOT, Donald K., Capt., USAF
ROSENBERG, Alan B., Capt., USAF
ROZANSKI, Lawrence T., Capt., USAF
RUTHERFORD, Oliver H., Jr., Capt., USAF
SCHWELLER, William C., Capt., USAF
SENGELMANN, Samuel S., Jr., Capt., USAF
SEVITTS, James B., Capt., USAF
SHAAK, Thomas A., Capt., USAF
SIPE, Harold G., Capt., USAF
SISLEY, Richard N., Lt., USN
SITTINGER, Eugene F., Capt., USAF
SKIBA, Gerald A., Capt., USAF
SMITH, Charles E., Capt., USAF
SMITH, Donald G., Capt., USAF
SMITH, Robert F., Lt., USN
SNYDER, Hugh P., Lt., USN
SNYDER, William G., Lt., USN
SODERSTROM, Ernest P., Capt., USAF
SONNLEITNER, Eugene T., Lt., USN
SPARKS, Rex M., Capt., USAF
SPEARS, Charles E., Capt., USAF
SPECK, Morton, Lt., USN
SPERBER, Norman D., Lt., USN
STALVEY, Arman D., Capt., USAF
STANCIL, Arthur C., Capt., USAF
STARSHAK, Thomas J., Lt., USN
STEARNS, Rexford H., Jr., Lt., USN
STEFIK, Leonard J., Capt., USAF
STEINBERG, Arnold D., Capt., USAF
STEVENS, Elmer J., Lt., USN
STEVENS, Otto O., Jr., Lt., USN
STUCKI, Grant A., Capt., USAF
TIMBERLAKE, Wayne, Capt., USAF
TOMICH, George, Capt., USAF
TUCKER, Bill E., Capt., USAF
WAGNER, Charles F., Capt., USAF
WAGNER, Norman E., Capt., USAF
WALKER, Daniel G., Capt., USAF
WEBB, Ernest J., Capt., USAF
WEISS, David E., Capt., USAF
WHITE, John P., Jr., Capt., USAF
WHITE, John W., Capt., USAF
WHITMAN, Jack D., Capt., USAF
WILLIAMS, George R., Capt., USAF
WILLIAMS, James F., Capt., USAF
WOLFE, Charles H., Capt., USAF
WOOD, Eason K., Jr., Capt., USAF

OFFICERS CERTIFIED BY SPECIALTY BOARDS

Supplementary Listing

According to current information from the Offices of the Surgeons General of the three military medical services, the following 20 officers have been certified by the boards indicated, since the listings published in successive issues of this *Journal* from October 1953 to April 1955.

Dental Corps

American Board of Periodontology

Charles F. Sumner, III, Capt., USA

American Board of Oral Pathology

William G. Sprague, Capt., USAF

American Board of Oral Surgery

Ogden M. Frank, Lt. Col., USA
Harvey S. Johnson, Capt., USN
William B. Johnson, Capt., USN
Winlaw A. Priebe, Col., USA

American Board of Prosthodontics

John P. Christensen, Lt. Col., USA
Perry W. Bascom, Lt. Col., USA
Walter J. Demer, Capt., USN
Oren H. Gaver, Lt. Comdr., USN
William E. Gullett, Capt., USN
James B. Lepley, Comdr., USN
Robert B. Lytle, Capt., USN
Kenneth D. Rudd, Maj., USAF

Veterinary Corps

American College of Veterinary Pathologists

Daniel P. Sasmore, Lt. Col., USAF

American Board of Veterinary Public Health

Kenneth F. Burns, Lt. Col., USA
Philip R. Carter, Col., USA
Charles H. Snider, Lt. Col., USAF
Mervyn B. Starnes, Col., USA
Albert A. Taylor, Lt. Col., USAF

OFFICIAL DECORATIONS

LEGION OF MERIT

Donald D. Flickinger, Brig. Gen., USAF (MC)

COMMENDATION RIBBON

Walter H. Bird, Col., USAF (DC)
John A. Durany, 1st Lt., MSC, USA
Carroll D. Goon, Capt., MC, USA
Frank E. Hicks, Jr., 1st Lt., MSC, USA
Howard R. Lawrence, Lt. Col., USAF (MC)
John E. Roberts, Col., USAF (MC)*
John K. Sitzman, Col., USAF (DC)

*First oak leaf cluster

FIRST INTER-AMERICAN CONFERENCE ON OCCUPATIONAL MEDICINE AND TOXICOLOGY

The first Inter-American Conference on Occupational Medicine and Toxicology will be held jointly by the University of Miami School of Medicine and the University of Havana School of Medicine 3 to 7 September 1956 in Miami. This year the University of Miami School of Medicine will serve as host. General Chairman is Dr. Homer F. Marsh, Dean of the University of Miami School of Medicine. The official language of the program will be Spanish.

Speakers from Venezuela, Mexico, Peru, Colombia, Chile, Puerto Rico, Cuba, and the United States will present papers on such topics as "A Medical Department in Industry," "Work and Fatigue in Industry," "Effects of Environmental Conditions on the Health of the Occupational Worker," "Recording of Medical Case Histories," "Occupation and Heart Disease," "The Treatment of Intoxications by Organic Phosphates," "Management of Berylliosis," and "Treatment of Manganese Intoxications."

Reviews of Recent Books

THE ROLE OF ALGAE AND PLANKTON IN MEDICINE, by *Morton Schwimmer*, M. D. and *David Schwimmer*, M. D., F. A. C. P. 85 pages. Grune & Stratton, Inc., New York, N. Y., 1955. Price $3.75.

This is an unusual book. In a mere 65 text pages the authors have reviewed an extensive literature (with actual reference to 312 publications) on the subject of the medical aspects of algae and plankton. These simple forms of life encompass diatoms, unicellular flagellates, Chlorella, shrimplike Crustacea, seaweeds, et cetera. They have been used in various ways in medicine for thousands of years. Current interest has been revived on the basis of the potentially enormous food supply available to mankind from the ocean. Among other medical aspects discussed are the allergic dermatitides, gastroenteritides, certain shellfish poisonings, and problems of water supply and sewage disposal. Although the subject matter is unfamiliar to most readers, this field does cross specialty interests and will contact almost every aspect of medical practice. The authors have provided us with a stimulating survey of a matter which will undoubtedly grow rapidly in importance.—*S. O. WAIFE, Lt. Comdr., MC, USNR*

BASIC SURGICAL SKILLS, A Manual with Appropriate Exercises, by *Robert Tauber*, M. D., F. A. C. S. 75 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1955.

This book presents a number of basic surgical maneuvers (knot-tying, suturing, et cetera), most of which are illustrated by line drawings. Purchase of the book entitles the buyer to a "training board" without additional charge. The "training board" is a 11½- by 12-inch board of pine wood to which can be attached hooks, nails, rods, rings, and gauze pads to aid in actual practice of the maneuvers presented in the book. The descriptions are concise and clearly presented. The line drawings are excellent and the multiple-figure sequences are easy to follow.

Although the concept of the presentation of basic surgical maneuvers accompanied by actual training exercises is to be commended, there are certain deficiencies in the book which must be noted.

A book of this sort should either present virtually all the accepted basic surgical maneuvers of all the surgical specialties or it should present a representative group of the most commonly used maneuvers. This book does neither. Instead it presents an assortment of surgical maneuvers which doubtless have served the author well but which are not necessarily representative of surgical technic in general.

For example, the method given for tying the so-called "slow" square knot (two-handed square knot) appears to be merely a modification of

the one-handed or "fast" square knot. Such a method would appear to be unsuitable for tying sutures under tension. Some evidence to support this view is given by the fact that the author devotes two pages and two figures to describing the use of a "knotholder" clamp which "prevents the knot from getting loose during the second half of the tying maneuver." In the reviewer's experience and observations, such a method for tying under tension is rarely used. One of the conventional two-handed square knots, which assures better control of the suture, would seem more appropriate if only one method is to be presented.

Throughout the book, one recognizes technics that are used particularly by gynecologists and obstetricians but that are not commonly used by surgeons of other specialties. If the book is to bear out its title, it should give proper representation to the technics of all the surgical specialties.

The need for a book of this sort is recognized, and the idea of practical exercises on the "training board" is heartily endorsed. With appropriate revisions, the book can be highly recommended for anyone anticipating a career in any field of surgery. As such, it might make a worthwhile addition to the curriculum of the undergraduate medical student.

—*JAMES H. STEWART, Lt. Comdr., MC, USN*

CLINICAL ANALGETICS, by *E. G. Gross*, Ph. D., M. D., and *M. J. Schiffrin*, Ph. D. American Lecture Series, Publication No. 273, A Monograph in American Lectures in Pharmacology, edited by *Chauncey D. Leake*, Ph. D. 101 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $3.

This volume is a readily usable work on the commonly used analgetics. The authors state the purpose of the monograph is to provide a concise, practical guide on analgetics for the general practitioner, dentist, pharmacist, and medical student. Short descriptions of the commonly used agents include salicylates, opiates, opioids, opiate antagonists, and local anesthetics.

A bibliography follows each chapter for further investigation of the agents described. Tables are given of commonly met proprietary preparations and the contents of such preparations.

Though the material in this monograph is not as extensive or as complete as found in standard textbooks on pharmacology, and the concepts presented are new in few instances, it is useful as a reference.

—*WILLIAM S. GEORGE, Col., MC, USA*

HYPOTHERMIC ANESTHESIA, by *Robert W. Virtue*, M. D., Ph. D. A Monograph in American Lectures in Anesthesiology, edited by *John Adriani*, M. D. 62 pages. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $2.50.

This monograph is a comprehensive review of the experimental and clinical application of hypothermia. The title, however, is misleading. As written, the reader would expect to read about the problems of

administering anesthetics to patients whose body temperature is to be lowered for certain surgical procedures. Actually, only a few pages are devoted to this facet of the general problem of hypothermia. The remainder of the monograph is concerned with a review of published reports and the author's personal experience with the use of cold hypothermia in medicine.

The monograph is short, concise, and informative. It is an excellent reference for source material for review and is highly recommended for both surgeon and anesthesiologist. — *JAMES G. KURFEES, Capt., MC, USN*

INTRODUCTION TO OPERATING-ROOM TECHNIQUE, by *Edna Cornelia Berry*, R. N., and *Mary Louise Kohn*, R. N., M. N. 154 pages. The Blakiston Division, McGraw-Hill Book Co., Inc., New York, N. Y., 1955. Price $4.

This timely and well-written textbook for students in operating-room technic will also serve for training nonprofessional personnel and as a review for the graduate nurse.

Sufficient background in surgery is included to make interesting but not time-consuming reading. The description of special equipment is adequate. Either basic technics and required equipment are fully described, or suitable references are given at the end of each chapter. The questions and assignment at the end of each chapter place emphasis on the important points. Pre- and post-operative care of the patient which is important and often omitted in books on operating-room technic is briefly discussed.

The author stresses the fact that anything that hangs over the edge of a sterile table should not be used as sterile; however, the illustration of instruments in a sterile setup shows them hanging over the edge of the table. This is not consistent with the technic advocated by the authors. Some additional points should have been stressed as not good technic, such as the use of the hot plate for sterile solutions in the operating room and the use of pins in sterile supplies.

The interesting chapter on radium will be of value to the reader. The one on anesthesia is an important addition to the book and is adequately covered; however, the chapter on medicolegal aspects is very brief and does not seem emphatic enough.

The print of the book is clear and easy to read, and the illustrations are effective. I would highly recommend this book as a textbook for teaching operating-room technic.

—*EDITH L. SHUTT, Maj., ANC, USA*

COMPLICATIONS OF REGIONAL ANESTHESIA, Etiology, Signs and Symptoms, Treatment, by *Daniel C. Moore*, M. D. 291 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $10.50.

This book is essentially an elaboration of the material which is found not only in this author's earlier text, *Regional Anesthesia*, but also in

the several excellent texts on this subject now available. It is an excellent reference for a teaching program but seems a little too expensive for its value to the practicing anesthesiologist. The book offers a fine collection of references at the end of each chapter which makes it even more desirable for a teaching library. The text is somewhat repetitive and more space is given to complications of the very rare blocks than seems necessary. By condensing some of the chapters, the book might be made cheaper and thus become a must book for all anesthesiologists. The illustrations, as usual with this author, are superb, and their reproduction by the publisher excellent.— *DAVID E. MacQUIGG, Maj., MC, USA*

RADIOGRAPHIC ATLAS OF SKELETAL DEVELOPMENT OF THE KNEE, A Standard of Reference, by *S. Idell Pyle*, Ph. D. and *Normand L. Hoerr*, Ph. D., M. D. 82 pages; 29 plates. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $4.25.

This atlas is the second in a series of several radiographic standards of reference for skeletal maturation, based on studies of human growth and development. The studies were begun by the late T. Wingate Todd and were a project of the Brush Foundation and the Western Reserve School of Medicine, Cleveland, Ohio. Several thousand children were studied. The first atlas in the present series was the *Radiographic Atlas of Skeletal Development of the Hand and Wrist*, by Greulich and Pyle.

There are several preliminary chapters describing how the plates were selected so that one series of standard plates is equally applicable for a boy or a girl. The principal part of the atlas consists of 29 reproductions of films of healthy children from birth to age 18. Key osseous features illustrating the current stage of development at a designated skeletal age are described and labeled on each plate. The authors point out that by standardizing these osseous features of the knee according to a moderate rate of skeletal development, either a fast rate or a slow rate of osseous development of the knee is thereby automatically indicated.

The text is well written and the reproductions are excellent. The atlas should be of considerable interest to radiologists, pediatricians, and investigators interested in a standard of reference for the maturation of the knee joint.— *JOHN L. HATCH, Capt., MC, USN*

FUNCTIONAL OTOLOGY, The Practice of Audiology, by *Morris F. Heller*, M. D., with *Bernard M. Anderman*, M. A., and *Ellis E. Singer*, M. A. 225 pages; illustrated. Springer Publishing Co., Inc., New York, N. Y., 1955. Price $5.50.

The author's description of a sound-treated room and equipment lacks detail that would enable one to set up a hearing testing facility without further information. The chapter on history taking is excellent and reflects the great experience of the author. The explanation of audiometric technic is excellent; descriptions of the technic of special

tests such as recruitment binaural and monaural, loudness balance, and difference limen are given in simplified terms.

The text is well organized and covers the field of audiology from history taking to rehabilitation. Generally speaking, the material is presented simply and is readily understandable. The author's description of the clinical aspects of diagnosis and appraisal of the patient as a whole shows a remarkable insight.

It is felt that this book will have its greatest value for audiometric technicians, audiology students, and physicians who are interested in diagnosis and rehabilitation of the hard of hearing; however, the material has not been covered in enough detail to be used as a reference.—*RALPH N. KRAUS, Lt. Col., USAF (MC)*

AN INTRODUCTION TO HUMAN ANATOMY, by *Clyde Marshall,* M. D. 4th edition. Revised by *Edgar L. Lazier,* Ph. D. 420 pages; 329 illustrations, 15 in color. W. B. Saunders Co., Philadelphia, Pa. 1955.

Although it is nowhere clearly stated, one gains the impression that this book is intended for the arts and science college student. This is the fourth edition in a period of 20 years. According to Dr. Lazier, "the most extensive revision" has been in the treatment of the neurosensory system. The two chapters on this subject constitute almost one fourth of the book.

The illustrations are usually good and are taken in the main from standard textbooks of anatomy. At various points paragraphs entitled "practical considerations" are inserted in the text. Footnotes dealing generally with etymology are liberally interspersed and should be helpful to the novice.

While the order of presentation may vary, the topics covered are conventional. One may again learn the derivation of the various coverings of the testicle acquired during its descent through the abdominal wall. However, there was no mention of the relationship of the inferior epigastric artery to the abdominal inguinal ring. In other sections taken at random, I was surprised to find practically no reference to the coronary arteries and certainly there exists some question as to whether the appropriate way to control venous hemorrhage is by compression on the "distal side."

The physician and the student of any of the sciences closely allied to medicine will find little or no use for this elementary presentation.

—*WILLIAM D. TIGERTT, Lt. Col., MC, USA*

THE MANAGEMENT OF OBSTETRIC DIFFICULTIES, by *Paul Titus,* M. D., revised by *J. Robert Willson,* M. D., M. S. 5th edition. 737 pages; with 348 text illustrations and one color plate. The C. V. Mosby Co., St. Louis, Mo., 1955. Price $12.50.

The fifth edition of Titus' *Management of Obstetric Difficulties* has been brought up to date in every section and chapter. It is very easy to

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read and contains much valuable information of considerable interest to student, practitioner, and specialist alike. Dr. Willson has definitely improved the entire text by including considerable new material and by deleting old, outmoded passages. He has called upon his depth of experience to accomplish this end, and one cannot help but heartily approve of his over-all policy of conservatism, especially when discussing controversial issues.

The first section deals exclusively with the problem of infertility and covers all aspects of diagnosis and treatment now available. The section on complications of labor is especially well presented and contains all the most modern concepts concerning this ever-present problem. The many photographs and drawings go a long way in explaining certain difficult passages in the text.

This is an excellent and timely revision of a classic obstetric text and in my opinion will be of inestimable value as a source of reference to anyone practicing obstetrics.—*WILLIAM S. BAKER, Jr., Capt., MC, USN*

THE TREATMENT OF RENAL FAILURE, Therapeutic Principles in the Management of Acute and Chronic Uremia, by *John P. Merrill,* M. D. 238 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., 1955. Price $6.75.

This is a well-written treatise by a recognized authority in the field of renal disease. The discussions of the physiology, pathology, and symptomatology of kidney disease in successive chapters give a particularly clear understanding of the pathogenesis and recognition of renal failure. Detailed management of both acute and chronic kidney disease is presented concisely enough to serve as a ready reference for the case at hand. Finally, treatment of renal failure by extra renal routes (the artificial kidney being the most prominent) is discussed.

After carefully reading this little book the writer was fully convinced that it serves its purpose as a guide to the treatment of renal disease. Those dealing with such problems will undoubtedly agree.

—*JOHN B. MacGREGOR, Capt., MC, USN*

REHABILITATION OF A CHILD'S EYES, by *Richard G. Scobee,* M. D., F. A. C. S. Revised by *Herbert M. Katzin,* M. D., F. A. C. S. 2d edition. 133 pages; illustrated. The C. V. Mosby Co., St. Louis, Mo., 1955. Price $2.85.

This little book of 133 pages is dedicated to the memory of Dr. Richard G. Scobee, a recognized authority in the field of extraocular muscles, who wrote the first edition appearing in 1949. Dr. Scobee died in 1952, and Dr. Herbert M. Katzin has revised and brought the book up to date, adding several illustrations.

The worried parents of the squinting child have many questions to ask. Why do the child's eyes deviate? Are the parents responsible for the condition? Will the child outgrow it? What is the effect of crossed

eyes on the child's personality? What is the proper treatment and when should it begin? What is the role of glasses and may they be discarded after operation? This book answers these and many more questions in terms which the layman can comprehend.

Simple illustrations are used to explain accommodation, fusion, suppression, the accommodation-convergence relationship, the various refractive errors, the types of squint, operative procedures, et cetera. A glossary is appended to explain the terms used.

Although this book is written primarily for the parents, it should be of particular interest also to physicians, other than those dealing with diseases and surgery of the eyes, who are in need of information which will enable them to give sound advice on the subject when consulted by the parents of the cross-eyed child. Certainly, every ophthalmologist doing extra-ocular muscle surgery would do well to have copies available for loan to parents.—*KARL J. PALMBERG, Capt., MC, USN*

THE BACK AND ITS DISK SYNDROMES, Including Injuries, Diseases, Deformities and Disabilities, with Notes on The Pelvis and Coccyx, by *Philip Lewin,* M. D., F. A. C. S., F. I. C. S. 2d edition, thoroughly revised. 942 pages; 371 illustrations and 4 color plates. Line drawings by *Harold Laufman,* M. D. Lea & Febiger, Philadelphia, Pa., 1955. Price $18.50.

The author has changed the title of this second edition from the earlier "Backache and Sciatic Neuritis" in order to emphasize the importance of intervertebral disk lesions among the causes of low back pain and sciatica. The book is encyclopedic in its presentation of the numerous and varied conditions which may give rise to backache with or without radiation of pain to the extremities.

The immensity of the subject is adequate reason for any fault in continuity and organization which may be apparent. There is considerable repetition, and the book could readily be condensed with no loss of context. Indeed there are some subjects, such as treatment of burns of the back and detailed operative procedures for leg equalization, that have questionable bearing on the title subject.

There can be little criticism of the material, which is for the most part clearly presented. Those seeking a ready reference for the diagnosis and treatment of back disorders, especially the conservative management, will find it in this comprehensive volume. It is rather a pity, however, that many old concepts such as "railway spine," "Kummel's syndrome," and the importance of congenital lumbosacral anomalies in the etiology of low back pain have been retained in this edition. It is also unfortunate that the author recommends primary closure of back wounds sustained under conditions of warfare, provided they are treated early and properly débrided. This does not conform to the existing military policy of delayed wound closure and might be a source of confusion to those called upon to perform emergency operations in the field.

—*ERNEST A. BRAV, Col., MC, USA*

CLINICAL ROENTGENOLOGY, Volume III, The Lungs and the Cardiovascular System Emphasizing Differential Considerations, by *Alfred A. deLorimier,* M. D., *Henry G. Moehring,* M. D., and *John R. Hannan,* M. D. 512 pages; 760 illustrations (5 in color). Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $20.50.

This, the third of four volumes of "Clinical Roentgenology," concerns the thorax and its contents. There are separate sections devoted to mediastinal tumor, congenital malformations of the heart and great vessels, acquired heart disease, cardiac trauma, cardiac failure, tumors and cysts of the heart and pericardium, and pericarditis.

Each section begins with a concise discussion of general, embryologic, anatomic, roentgenologic, and technical considerations. Subdivisions of each section likewise contain introductory paragraphs of pertinent information concerning particular anatomic areas and pathologic conditions. The section on the lung is the largest and it presents the numerous pulmonary and bronchial disorders associated with inflammation, neoplasia, and hazardous occupation. This chapter is particularly well organized and contains a great deal of case material. Other sections are equally well written and the discussion of each subject includes a brief summary of clinical and laboratory data necessary for understanding and diagnosis.

The format is excellent. The illustrations are well chosen and of excellent quality. The table of contents is detailed and complete. The index is excellent and the bibliography adequate.

The great number of illustrations might place the book in the category of an atlas, yet it should prove to be both a text and reference book for the student and the experienced radiologist.

—*ELMER A. LODMELL, Col., MC, USA*

NEUROGLIA, Morphology and Function, by *Paul Glees,* M. A., D. Phil., M. D. American Lecture Series, Publication No. 260, A Monograph in American Lectures in Neurology. Edited by *Charles D. Aring,* M. D. 111 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $5.

This monograph is a well-organized and well-written presentation of past and present concepts on neuroglia. The author stresses the dynamic aspects of neuroglial function, pointing out that their function may extend beyond the protection, support, and insulation of the neurones, and presents evidence suggesting that neuroglia may have an influence directly related to neuronal metabolism and synaptic activity.

The book includes considerable historical background on the various and successive concepts of the nature and functions of neuroglia that have been held over the past years by various observers. After a short comparative anatomic résumé, the main body of the monograph is concerned with the morphologic and functional studies of neuroglia and their reactions to pathologic and functional conditions. The extensive bibliography of almost 300 references is a valuable compilation of data

on the subject of neuroglia, covering recent and past references in the European and American literature in journals of anatomy, pathology, neurology, and physiology. The 49 illustrations include 8 photomicrographs and 41 drawings.

By the nature of the subject and its treatment, this book will be of interest and value only to a limited group, including neurohistologists, neurophysiologists, and biochemists working in the field of neurophysiology. Its value to those not working in or not very close to the field of neurologic research is problematical.

—NELSON S. IREY. Lt. Col., MC, USA

THROMBOEMBOLIC DISEASE, by *Geza De Takats*, M. D., M. S., F. A. C. S. Edited by *Michael E. De Bakey*, M. D., and *R. Glen Spurling*, M. D. 55 pages. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $2.25.

This monograph on thromboembolic disease, one in the series of the American Lectures in Surgery, presents a concise discussion on the methods of therapy employed on the author's service. The monograph is subdivided into three main sections, (1) clinical forms of thromboembolism, (2) prevention of thromboembolic disease in surgical practice, and (3) methods of treatment. Of particular interest is the author's clear differentiation of venous thromboses of the lower extremities into 4 types. In the section on therapy the author vigorously advocates the employment of heparin as an anticoagulant and deprecates the frequently inadequate and incomplete therapy utilizing the coumarins. A waning enthusiasm for peripheral vein ligations is expressed.

This monograph is readable and free of the usual confusing terminology so often encountered in the field of intravascular clotting. It should be most useful to the clinical surgeon. A rather complete bibliography is included for those interested in further reference.

—HERRICK M. THOMAS, Maj., USAF (MC)

THE MECHANISMS OF HEALING IN HUMAN WOUNDS, A Correlation of the Clinical and Tissue Factors Involved in the Healing of Human Surgical Wounds, Burns, Ulcers, and Donor Sites, by *Shattuck W. Hartwell*, M. S., M. D., Ph. D. (Surg), F. A. C. S., F. I. C. S. 166 pages; 44 illustrations. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $4.75.

This monograph is written by a clinical surgeon who for many years has taken a special interest in the study and observation of the mechanisms involved in the healing of human wounds. Here he summarizes his conclusions and those of other observers. A complete bibliography of the subject is also included.

The volume is detailed enough to give the reader a useful knowledge of the mechanism involved in wound healing. At the same time it is brief enough and written in such an interesting manner that it can be absorbed in one evening. Special attention is given to the healing of human wounds in contrast to healing in the wounds of experimental

animals, and the differences and similarities are clearly enunciated. Various histologic types of tissue are dealt with individually in regard to the method of healing. Photomicrographs illustrate such interesting phenomena as the ameboid extension of epithelial cells to form a membranous covering of the epithelial wound and the infiltrating lymphocytes changing to macrophages in healing of wounds. Schematic drawings are used to demonstrate other features of wound healing.

Basing his discussion on the underlying physiologic and physical factors involved, the author points out how surgical technic may play a deciding role in the effectiveness of healing, and when it is to be considered chiefly primary or secondary. The final chapter presents "a new concept of the basal cells of human epithelium related to carcinoma." It serves to stir the reader's imagination as he reviews the author's concept of the mechanism of wound healing.

This publication is extremely stimulating reading and gives a clear-cut concept of human wound healing and its clinical significance.

—*EMMETT F. NORWOOD, Capt., MC, USN*

THE PATHOGENESIS OF POLIOMYELITIS, by *Harold K. Faber,* M. D. 157 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $5.

This book was written to clarify concepts on the basic nature of poliomyelitis. Much of the review is based on experiments conducted by the author and his associates and on a series of papers published since 1940. The author comments that some of his observations are published for the first time in this book.

The publication of this review is particularly timely in view of the recent interest in immunization against poliomyelitis. The perennial controversy as to whether the virus is strictly neurotropic or has affinities for other than nervous tissue is discussed. The author favors the concept of primary neurotropism. The controversy over the portal of entry of the virus is also presented.

The author frequently summarizes his discussions and keeps the reader from getting lost in some of the theoretical discussions. At the end of the book the author reviews his conclusions in a recapitulation of the material. The material is well organized and there is an extensive bibliography. Neurologists, internists, and pediatricians will find this a helpful and up-to-date review of current knowledge and concepts about poliomyelitis.—*ROBERT L. WILLIAMS, Maj., USAF (MC)*

A HANDBOOK OF HOSPITAL PSYCHIATRY, A Practical Guide to Therapy, by *Louis Linn,* M. D. 560 pages. International Universities Press, Inc., New York, N. Y., 1955. Price $10.

This volume is not a textbook of administrative psychiatry in the usual sense and has little to do with the logistics of hospital management. Rather, it is a practical, clearly written, absolutely sound and

dynamic exposition of the proper functioning of the human and material elements of the therapeutic community, the center of which is the mental hospital. The book deals with such matters as individual and group therapy in a mental hospital setting and the place, function, and interrelations of the psychiatrist, psychologist, social worker, nurse, chaplain, recreational and occupational worker, and even the dietitians and the volunteer worker. It contains excellent discussions of the influence of hospital architecture and décor, administrative procedures, staff education, outpatient and family care, and the role of the patient's relatives and his community in forwarding the over-all objective of patient rehabilitation.

One theme of this valuable book is the necessity for an *integrated,* holistic approach to the goal of effective treatment in which every aspect of the hospital organization is essentially participant. One of the larger values of this book is the broad, informed picture it affords for *all* members of a hospital organization of the desirable methods and functions of the individual units and ancillary services that comprise their institution. In addition, this is a handy reference book containing a wealth of practical information of particular interest to the hospital psychiatrist concerning such matters as the special problem of the addict, the criminal insane, forensic psychiatry, the sexual deviate, clinical statistics, and hospital research problems. The excellent bibliography includes a brief précis of the content and approach of each reference cited.—*CHARLES S. MULLIN, Capt., MC, USN*

HYPNOTIC SUGGESTION, Its Role in Psychoneurotic and Psychosomatic Disorders. A Thesis, by *S. J. Van Pelt,* M. B. 95 pages; illustrated. Philosophical Library, New York, N. Y., 1956. Price $2.75.

This book, written by the President of the British Society of Medical Hypnotists, is short, concise, and so well written that even a nonmedical reader could understand the subject of hypnosis after spending a few hours with the book.

The author refers to this work as a thesis. In this thesis he gives a short historical outline of hypnotism, the nature of the hypnotic state, and methods of inducing hypnosis. He rightfully denounces the demonstration of hypotism on the stage.

In this thesis the author claims originality for three findings. The first is that hypnotism is a concentration of the mind. He explains this well, states that a light hypnotic state may be obtained in 95 per cent of people. The second original theory is that hypnotic suggestion plays a role in the etiology of psychoneurosis. He states that by hypnotic suggestion a normal person may develop a condition indistinguishable from a geniune psychoneurosis. The third finding is an original method of treating psychoneurosis by hypnosis in a few visits. In the first visit a history is taken, in the second an explanation of what is to be accomplished is given to the patient, and light hypnotic sleep is in-

duced. In the third visit he again is placed in a light sleep, then informed that within a week a thought will enter his head which will show him why he first became frightened. The author states that invariably during the week the thought will appear and at the fourth session the patient will relate the incident which is the cause of his illness. The explanation for his illness is then imparted to him. The acutal number of visits required may be four or, in difficult cases, as many as 12. The treatment consists of relaxation, realization, and re-education, and 12 case histories are presented.

—FRANCES L. WILLOUGHBY, Comdr., MC, USN

EXPERIMENTAL PSYCHOLOGY, A Series of Broadcast Talks on Recent Research, by *A. J. Watson, Harry Kay, J. A. Deutsch, B. S. Farrell, Michael Argyle,* and *R. C. Oldfield.* Edited by *B. A. Farrell.* 66 pages. Philosophical Library, New York, N. Y., 1955. Price $2.75.

An American reader accustomed to the scholarly, critical, detailed reporting of experimental psychology such as that done by Woodworth or Stevens is apt to respond with disappointment to this book. The subtitle is misleading and might better be something to the effect of "brief discussions of selected research from selected areas of psychological research."

As indicated by the subtitle, the contents consist of six radio addresses delivered over the British Broadcasting System. The areas from which the talks are chosen include perception, adult learning and memory, motivation, some psychoanalytic hypotheses, and social behavior. The concluding address attempts to set forth some of the problems confronting researchers in the psychology of behavior, including potential lay reactions to their findings.

Although the volume does not merit the title "experimental psychology" and is not likely to attract the attention of the lay audience for whom it was apparently intended, its initial objective—the promotion of a better understanding of what psychologists are interested in and how their objectives are achieved—merits approval. American psychologists might well consider similar programs.

—JAMES W. LAYMAN, Maj., MSC, USA

UNDERSTANDING PEOPLE IN DISTRESS, Emotional and Mental Disorders, Their Cause, Care, and Cure, by *Barney Katz,* Clinical Psychologist, and *Louis P. Thorpe,* Professor of Psychology. 357 pages. The Ronald Press Co., New York, N. Y., 1955. Price $4.

The purpose of this introduction to neuropsychiatric problems is to explain the cause, care, and cure of mental and emotional disorders in simple language and for reading by the average man. Nevertheless, professional workers in the field of human maladjustment will find the volume useful for rapid review and occasional reference in matters of psychopathology. The writers perform an exacting job in intelligible and thorough fashion.

The approach is chiefly of the psychoanalytic order. However, the authors are inclined to ascribe the causation of practically all emotional disorders to early acquired feelings of insecurity and inadequacy on the part of the sufferer. They present the entire gamut of neurologic and psychiatric problems in tabloid style, illuminated by short, typical case histories. One would like to know more about the technic of psychotherapy pursued by the authors, as their case histories all eventuate in "cure" or great relief.

As to be expected with a popular work on psychiatry, their pronouncements are often dogmatic and sometimes questionable. For instance, they hold that only Huntington's chorea has been accepted by scientists as a hereditary mental disorder. They neglected to mention, for example, amaurotic familial idiocy or phenylpyruvic oligophrenia. Again, they state, "The secure person is one who has received as a child complete, continuous, and unqualified acceptance by his parents." But many early orphans have no doubt grown up into secure and happy adults by virtue of other felicitous interpersonal experiences.

The format is handy and the type readable. A selected bibliography and concise index enhance the utility of the volume.

—*ARTHUR J. ORANGE, Lt. Col., MSC, USA*

AN ELEMENTARY TEXTBOOK OF PSYCHOANALYSIS, by *Charles Brenner*, M. D. 219 pages. International Universities Press, Inc., New York, N. Y., 1955. Price $4.

In psychiatry the yearly yield of books and papers is overwhelming, and the plethora has tended to be increased by the publication of reviews, annuals, surveys, and yearbooks as well as volumes presenting an outline of, the foundations of, the essentials of, a synopsis of, the fundamentals of, or the introduction to a particular area of psychiatry or a related field. At first glance it appeared that this book was another in the last category. A reading proves otherwise. It is not a résumé for psychiatrists or trained professional workers in related fields. In his introduction the author sets himself a goal in keeping with the title. This is to be an elementary textbook setting forth the fundamentals of psychoanalytic theory and providing an introduction to the literature of psychoanalysis for the physician, the psychiatrist, the psychologist, and the social worker. This the book proposes to do without demanding from the reader a previous psychoanalytic knowledge.

Dr. Brenner has admirably reached his goal. In eight chapters and only 199 actual pages of text, he has presented the core of basic psychoanalytic theory and fact with striking clarity. Many excellent clinical examples illustrate these principles, and where the use of unfamiliar psychoanalytic terms is necessary, great pains are taken to assure the reader's understanding. At the same time a historical sketch of the development of Freud's basic concepts and the changes and refinements in those concepts as they took place is woven into the presentation. Changes in the meaning of terms that took place as concepts were

altered are also marked out for the reader's guidance. Each chapter is supplemented by suggested references for further reading. An alphabetical reference list is included at the end of the book, as is a very adequate index.

Dr. Brenner has turned out a factual, clear, concise presentation of basic modern psychoanalytic thought. It is well named. It is a textbook. As such it is unique, and it will, no doubt, fill a very definite need in the training of medical students, interns, psychiatric residents, and psychoanalytic candidates as well as students of allied fields. It may also become a classic in the field of teaching the teacher how to make extremely technical and complicated material as clear and concise as possible without loss of substance. —*ROBERT E. SWITZER, Comdr., MC, USN*

ATOPIC DERMATITIS, edited by *Rudolf L. Baer,* M. D. 112 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., 1955. Price $2.50.

This is a small text the purpose of which, as stated by the editor, is to provide "comprehensive information on the many facets of this very important skin disease." Material was previously published in the *American Practitioner and Digest of Treatment* for July 1955. It traces the development of the concept of atopic dermatitis, its clinical appearance, and the confusion existing in its histologic picture. Sulzberger points out that it is responsible for 3 to 5 per cent of all discharges from the service for disease.

Pictures and charts are presented to illustrate the infantile, the childhood, the adolescent, and young adult phases of the condition and its differential diagnosis. Kierland discusses certain stigmata associated with the disease and Rostenberg explores the theories concerning its pathogenesis. Management of atopic dermatitis is presented by Sternberg and Newcomer. Nothing is offered in this section that is not common knowledge.

Of interest are the following points: (1) The sweat retention syndrome is important as an etiologic factor. (2) Benefit is frequently derived from a change of environment. (3) None of the cardinal features of the disease are altered by any psychiatric approach. (4) The opinion of Sulzberger that the so-called typical personality of the affected individual is a result of the disease rather than a contributing factor of the condition is discussed. (5) It is implied that the higher brain centers or the adrenal medulla are involved in the abnormal responses of the skin, and that research involving the autonomous nervous system and the higher brain centers is indicated to locate the true pathogenesis of atopic dermatitis. (6) The concept is presented that first there must be a suitable soil in a diathetic individual. Scratching of the skin in such a person leads to the cutaneous lesions.

It is possible that this small book may prove very worthwhile. It is questionable, however that a book should be published on every important dermatologic condition. With the growth and development of

dermatology the field is now big enough to warrant a textbook of dermatology with truly multiple authorship or even an encyclopedia of dermatology rather than a multitude of separate books.

—*WILLIAM N. NEW, Capt., MC, USN*

THE BLOOD-BRAIN BARRIER, With Special Regard to the Use of Radioactive Isotopes, by *Louis Bakay*, M. D., F. A. C. S. 154 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $5.50.

This monograph on the blood-brain barrier is a complete treatise on the subject, starting with its historical background, the possible anatomic locations of the blood-brain barrier, and its physiology and development. The author discusses his experiments with various radioactive isotopes, especially radioactive phosphate and its relation to the blood-brain barrier. The artificial changes in the permeability of the blood-brain barrier by such means as brain trauma, cerebral infection, toxins, x-ray radiation, allergic agents, cerebral embolism, thrombosis, hemorrhage, and contrast medium used in carotid arteriography are amply discussed.

A large section of the book is devoted to the difference in the uptake of radioactive isotopes by various cerebral tumors in contrast to the normal brain. This difference has been an aid in the localization of these tumors both before and at operation. The reviewer believes that a chapter on the permeability of the blood-brain barrier in the normal and infected brain to the various antibiotics and sulfa drugs commonly in use would increase its attractiveness to clinicians.

The book is concise, well written, and easy to read, and it has an extensive bibliography. It will be of interest mainly to neurosurgeons, neurologists, clinicians, and research workers who deal with brain metabolism.—*WILLIAM J. JAMES, Capt., MC, USN*

PUBLIC RELATIONS FOR THE PHARMACIST, by *William H. Hull*, M. A. 132 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., 1955. Price $4.50.

This book, written in an entertaining and highly readable fashion, is well organized and simply presented. For the pharmacist in civilian life it offers an explicit, workable public relations program. For the pharmacist in military service it offers little that he can apply to his everyday work.

Of all the sections, the one dealing with "Two of Pharmacy's Problems" is by far the most interesting because it discusses freely and frankly the problem which the pharmacist faces when the physicians who serve the people in his community are "dispensers" rather than "prescribers." This section also discusses frankly the ethics involved in any doctor-pharmacist relationship and suggests the means whereby both parties may retain their self-respect while working together to serve their community.

Although the book is both easy and fast reading, it contributes nothing new to the public relations field. General principles, however, are simply illustrated, so that even a specialist in the field can profit from the author's well-organized presentation. It is unfortunate that the text's references to certain illustrative "figures" in the book require that the reader must turn the pages back or forth frequently. Despite this shortcoming, however, it should prove of great value to the civilian pharmacist.— *WILLIAM L. AUSTIN, Maj., MSC, USA*

WORLD-ATLAS OF EPIDEMIC DISEASES, Part II, Second and Third Issues, edited by Professor Dr. med *Ernst Rodenwaldt,* under the Sponsorship of the Heidelberger Akademie Der Wissenschaften. Second issue, 38 pages; 10 colored maps. Third issue, 42 pages; 9 maps. Falk-Verlag, Hamburg 1, Germany, 1954. Price 57.50 Deutsche marks, each issue, plus extra charge for postage and packing.

The fine world-over epidemiologic studies of diseases continue to be produced with remarkable regularity. Each issue contains a number of diseases with statistic and clinical descriptions in English and German, and maps which are the hallmarks of this publication. The pages are not serially numbered but are designed to fit into the loose-leaf system begun in the original publication. The disease entities and maps contained in the second and third issues of volume 2 are listed. The second issue includes: paratyphoid A in Europe, diphtheria in Central Europe, diphtheria in Europe, dengue in the Mediterranean, yellow fever in Africa, trench fever in Europe, leishmaniasis in Africa, filariasis in Africa, distribution of population in the Mediterranean, and distribution of population in Africa. The third issue includes: scarlet fever, morbidity (world); scarlet fever, lethality (world); plague in Europe; plague in Africa; tularemia in Central Europe; louse-borne relapsing fever (world); tick-borne relapsing fever (world); Q fever in Europe; amebic dysentery (world); and ascariasis (world).

Due to availability of official Japanese government health statistics, the rates for the various diseases in Japan were reviewed. There were only minor variations from the maps noted; for example, scarlet fever morbidity indicated a lower morbidity for the years 1919 through 1953 than the rates indicated for the Japanese Islands on the World-Atlas map. The text clearly presents the difficulties in using statistics for the preparations of world maps because of variations in criteria for diagnosis, clinical changes in severity, and the purely mechanical classification systems in use in various countries.

For the epidemiologic student and expert alike, the detailed work and faithful, accurate mapping will make a most complete and authoritative reference. The publishing of the next part will be eagerly awaited by public health physicians and medical libraries throughout the world.— *GOTTLIEB L. ORTH, Col., MC, USA*

New Books Received

Books received by the *U. S. Armed Forces Medical Journal* are acknowledged in this department. Those of greatest interest will be selected for review in a later issue.

PULMONARY CARCINOMA, Pathogenesis, Diagnosis, and Treatment, edited by *Edgar Mayer,* M. D., and *Herbert C. Maier,* M. D. 18 contributors. 540 pages; 213 figures; 4 plates in color. Published by New York University Press, New York, N. Y., 1956. Distributed by J. B. Lippincott Co., Philadelphia, Pa. Price $15.

BING'S LOCAL DIAGNOSIS IN NEUROLOGICAL DISEASES, by *Webb Haymaker,* M. D., with chapters by *Richard G. Berry,* M. D., *Bernard S. Epstein,* M. D., and *Paul I. Yakovlev,* M. D. Translated, revised, and enlarged from the 14th German edition. 478 pages; 225 illustrations, including 9 in color. The C. V. Mosby Co., St. Louis, Mo., 1956. Price $16.75.

CULTURE AND MENTAL DISORDERS, by *Ralph Linton.* Edited by *George Devereux.* 139 pages. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $4.50.

COLLAGEN DISEASES Including Systemic Lupus Erythematosus, Polyarteritis, Dermatomyositis, Systemic Scleroderma, Thrombotic Thrombocytopenic Purpura, by *John H. Talbott,* M. D., and *R. Moleres Ferrandis,* M. D. 232 pages; 30 figures; 16 plates in color. Grune & Stratton, Inc., New York, N. Y., 1956. Price $6.50.

HEALTH OBSERVATION OF SCHOOL CHILDREN, A Guide for Helping Teachers and Others to Observe and Understand the School Child in Health and Illness, by *George M. Wheatley,* M. D., M. P. H., and *Grace T. Hallock.* Illustrations by *Barbara Pfeiffer.* 2d edition. 488 pages; illustrated. The Blakiston Division, McGraw-Hill Book Co., Inc., New York, N. Y., 1956. Price $6.50.

THE LEGACY OF SIGMUND FREUD, by *Jacob A. Arlow,* M. D. 96 pages. International Universities Press, Inc., New York, N. Y., 1956. Price $2.

THE YEAR BOOK OF NEUROLOGY, PSYCHIATRY AND NEUROSURGERY (1955-1956 Year Book Series). Neurology, edited by *Roland P. Mackay,* M. D. Psychiatry, edited by *S. Bernard Wortis,* M. D. Neurosurgery, edited by *Percival Bailey,* M. D., and *Oscar Sugar,* M. D. 576 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1956. Price $7.

ADRENAL FUNCTION IN INFANTS AND CHILDREN, A Symposium, edited by *Lytt I. Gardner,* M. D. 221 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., 1956. Price $6.75.

DYNAMICS OF PSYCHOTHERAPY, The Psychology of Personality Change. Volume I, Principles, by *Percival M. Symonds,* Ph. D. 210 pages. Grune & Stratton, Inc., New York, N. Y., 1956. Price $5.50.

LAUGHTER AND THE SENSE OF HUMOR, by *Edmund Bergler,* M. D. 297 pages. Intercontinental Medical Book Corp., New York, N. Y., 1956, in cooperation with Grune & Stratton, Inc., New York, N. Y. Price $5.

TUMORS OF THE SKIN, by *Herbert Conway,* M. S., M. B., M. D., American Lecture Series, Publication No. 270, A Monograph in The Bannerstone Division of American Lectures in Surgery, edited by *Michael E. DeBakey,* M. D., and *R. Glen Spurling,* M. D. Plastic Surgery Division, edited by *James Barrett Brown,* M. D. 267 pages; 178 figures; 3 plates in color. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $13.50.

ANGIOCARDIOGRAPHIC INTERPRETATION IN CONGENITAL HEART DISEASE, by *Herbert L. Abrams,* M. D., and *Henry S. Kaplan,* M. D. American Lecture Series, Publication No. 279, A Monograph in The Bannerstone Division of American Lectures in Pediatrics, edited by *John A. Anderson,* M. D. 233 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $11.75.

CONTEMPORARY PSYCHOTHERAPISTS EXAMINE THEMSELVES, An Evaluation of Facts and Values Based Upon Guided Interviews with Forty-three Representatives of Various Schools, by *Werner Wolff,* Ph. D. American Lecture Series, Publication No. 264, A Monograph in The Bannerstone Division of American Lectures in Psychology, edited by *Molly Harrower,* Ph. D. 300 pages. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $6.75.

THE YEARBOOK OF MODERN NURSING 1956, A Source Book of Nursing, edited by *M. Cordelia Cowan.* Foreword by *Mary M. Roberts.* 446 pages. G. P. Putnam's Sons, New York, N. Y., 1956.

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